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REGARDING MORPHOLOGICAL AND FUNCTIONAL CHANGES OF THE INTERNAL EAR IN ARTERIOSCLEROSIS.*

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This report is based upon a study of the microscopic sections and the functional records of hearing of 26 cases (52 ears) in the collection of the Otological Research Laboratory of Johns Hopkins University, supplemented by the general clinical histories and autopsy protocols. The clinical diagnosis of arteriosclerosis had been confirmed at autopsy in all of the cases selected for study. No cases were included in which there was a clinical history of syphilis or otitis media. In two cases the sections did reveal changes in the middle ear indicative of a healed otitis media, but in neither were these of a nature to have materially affected the hearing at the time of the functional examination. In one other case it was found that a mild otitis media had developed between the time of examination and death.

The ages of the patients ranged between 47 and 78 years; four were between 47 and 50, seven between 51 and 60, and 15 over

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CHART 1.—TABULATION OF OBSERVATIONS

Case No.	Age	Race	Sex	Condition of			Blood pressure	Condition of					Hearing loss in range			Note
				Internal carotid	Cerebral vessels	Meatus vessels		Organ of Corti	External sulcus cells	Stria vascularis	Spiral ganglion	Nerve fibres in spiral lamina	Low	Middle	High	
1.	50	W	M	+	+	—	250/170	+	+	?	—	—	—	—	+	
2.	50	C	F	+	+	+	245/170	—	—	—	—	—	—	—	—	
3.	47	C	M	+	+	+	156/110	?	—	—	—	—	—	—	+	
4.	44	C	M	—	—	—	115/70	—	—	—	—	—	—	—	—	
5.	53	C	M	+	+	+	125/70	+	+	+	+	+	—	—	—	Slight uniform loss for all tones
6.	58	W	M	+	+	+	110/58	—	—	—	—	—	+	+	+	
7.	55	W	F	—	—	+	123/70	—	—	—	—	—	+	+	+	
8.	53	W	M	++	?	+	144/90	—	—	—	+	+	+	+	+	
9.	52	W	M	*	—	+	148/88	+	—	—	—	—	—	+	+	
10.	54	C	M	++	+	+	300/110	—	—	—	?	—	+	—	?	Drops at C256 and C8192—otherwise good

LEGEND : W=white, C=colored, M=male, F=female, R=right, L=left, +=definite atrophy or loss, —=no definite changes from normal, ?=early changes or questionable changes, *=data not obtained.

CHART 1.—TABULATION OF OBSERVATIONS (Continued)

Case No.	Age	Race	Sex	Condition of: Internal carotid	Cerebral vessels	Meatus vessels	Blood pressure	Condition of: Organ of Corti	External sulcus cells	Stria vascularis	Spiral ganglion	Nerve fibres in spiral lamina	Hearing loss in range: Low	Middle	High	Note
11.	57	C	F	+	?	?	*	—	—	—	+	?	—	—	+	
12.	64	C	M	+	+	+	165/120	?	+	+	+	+	+	+	++	
13.	68	W	F	++	+	+	250/120	L+ R—	L+ R—	L+ R—	+	+	+	+	+	
14.	67	C	M	*	?	*	160/95	—	?	—	L+ R—	L+ R—	+	+	++	
15.	64	W	M	+	+	+	180/105	—	?	+	+	+	—	—	++	
16.	64	C	M	*	+	+	190/110	—	—	—	+	+	+	+	—	Uniform loss but high tone loss less than the average
17.	68	W	F	*	+	+	220/140	—	—	—	+	+	+	+	++	
18.	60	W	M	+	+	?	170/80	—	L+ R—	—	+	+	—	—	+	
19.	60	W	M	+	+	?	150/110	L+ R—	+	L+ R—	+	+	—	+	+	
20.	65	W	M	+	—	?	158/100	—	—	—	+	+	—	+	+	
21.	74	W	M	*	+	+	220/130	R+ L—	R+ L—	R+ L—	+	+	+	++	++	
22.	73	W	F	+	?	—	*	?	—	*	+	?	—	+	++	
23.	75	W	M	*	—	—	218/90	L+ R—	L+ R—	L+ R—	+	?	—	—	—	
24.	78	W	F	+	—	?	170/88	?	?	—	+	+	+	+	++	
25.	70	C	M	—	—	—	90/45	—	?	—	?	?	—	—	—	
26.	70	W	M	?	+	+	136/86	—	—	?	+	+	—	—	+	

LEGEND: W = white, C = colored, M = male, F = female, R = right, L = left, + = definite atrophy or loss, — = no definite changes from normal, ? = early changes or questionable changes, * = data not obtained.

60 years of age. The immediate causes of death, as stated in the clinical histories, were as follows: myodegeneration of the heart, 11 cases; cerebral hemorrhage, bronchopneumonia, pulmonary tuberculosis, carcinoma, each three cases; nephrosclerosis, lobar pneumonia, postoperative peritonitis, each one case.

Besides the changes in the larger vessels and in those of other organs there was also sclerosis of the internal carotid artery and of the cerebral arteries to be found in the majority of the cases. The blood pressure in most of these cases was above normal (see Chart 1).

Arteriosclerosis as a degenerative change in the walls of the arteries accompanies more or less the progressing age and it is observed to some degree practically in every person. Also that there

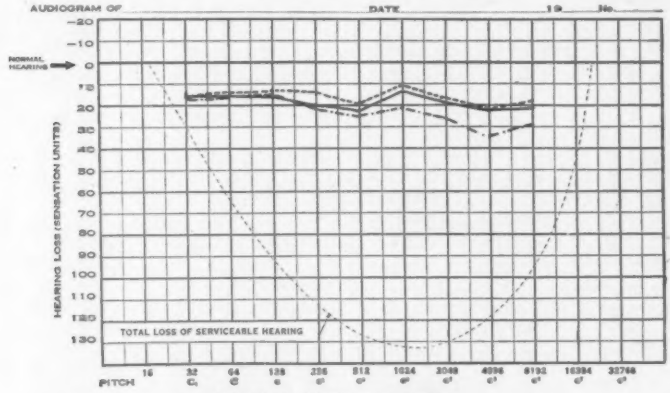

Chart 2. 41-50 year group.
——— Bunch's average curve of patients with arteriosclerosis.
- - - - - Bunch and Raiford's average curve.
-.-.-.-.- Average curve of the reported cases.

is a gradual loss of acuity for the higher tones (c^4 and above) with advancing age has been generally recognized since the work of Zwaardemaker (1893), when he coined the term, "presbyacusia." The amount and extent of this average hearing loss has been again studied recently by Bunch (1929) and Bunch and Raiford (1931). The observations of these authors are especially suitable for comparison with the cases I am reporting, inasmuch as the same instruments were used, and under the same conditions and by the same examiners. In the above papers attention was not given to clinical diagnosis; but in another paper Bunch (1931) reported the average hearing acuity of patients in the several decades of life with a clinical

diagnosis of arteriosclerosis. He found no significant difference in the hearing when compared to others without such diagnosis. It should be noted that he used in his study the records of patients who either did not die during hospitalization or for whom autopsy permission was not given; therefore, in only a few cases was the clinical diagnosis confirmed. This is a possible explanation of the fact that the average hearing in the cases I am reporting was poorer than found by Bunch. The curves of the average hearing in the cases here reported are contrasted to Bunch's and Bunch and Raiford's average curves for the corresponding age groups in Charts 2, 3 and 4 (see Charts 2, 3 and 4).

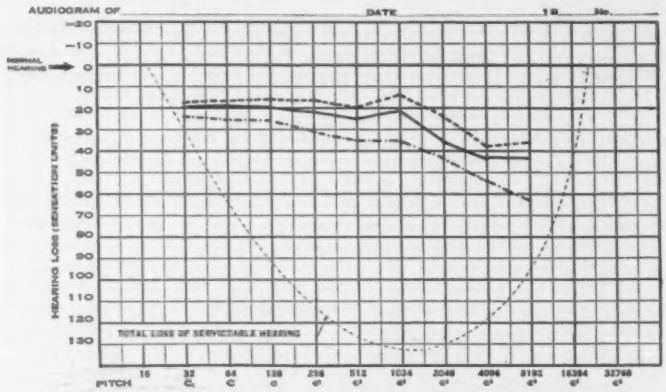

Chart 3. 51-60 year group. Part of these cases were examined with the Western Electric 2A Audiometer, where values were not obtained for C^1 (32 d. v.) and tones above c^6 (8192 d. v.), therefore averages were not obtainable.
——— Bunch's average curve of patients with arteriosclerosis.
- - - - - Bunch and Raiford's average curve.
-.-.-.-.- Average curve of the reported cases.

Although old age and typical high tone loss are related as a rule, we have seen several cases who had very little loss of hearing and the sections of the cochlea have not shown considerable changes either in the ganglion cells or in the organ of Corti. But in the majority of cases of marked arteriosclerosis the loss of hearing is greater than in persons with moderate arteriosclerosis and is usually accompanied by more or less atrophy of the spiral ganglion, the nerve fibres in the osseous spiral lamina and the organ of Corti of the basal turn.

From Chart 1 it can be seen that in the lower basal turn atrophic changes in the spiral ganglion and nerve fibres in the osseous spiral lamina are of more frequent occurrence than in the organ of Corti.

Postmortal changes and artefacts due to the method of histologic preparation are present in the microscopic sections studied, as in all human temporal bone material. Comparisons of these sections with many others, prepared by the same methods, but from cases in which the hearing was known to be good and in which arteriosclerosis was not present, constitute the basis of the judgment which I have had to use in the interpretation of the true significance of the things actually seen in the sections.

Atrophy of the organ of Corti in the lower basal coil was observed in seven cases (27 per cent), but in four of them the atrophy was only unilateral. In four of these there were definite

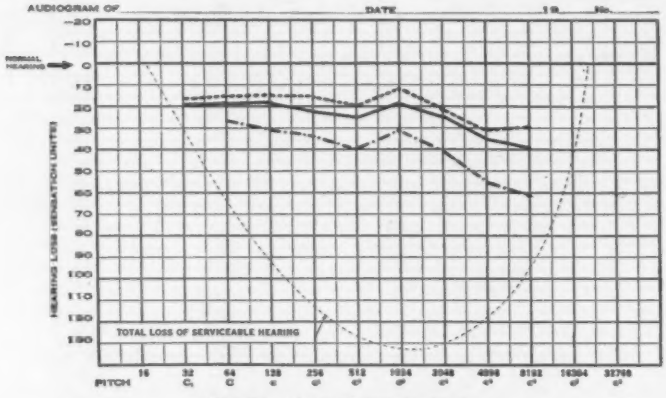

Chart 4. Over 60 year group.
——— Bunch's average curve of patients with arteriosclerosis.
- - - - - Bunch and Raiford's average curve.
-.-.-.-.- Average curve of the reported cases.

changes in the meatus vessels, and in one other questionable early changes. Appearances of early atrophy of the organ of Corti were seen in four cases (15.4 per cent). In two of these there were definite changes in the vessels of the internal meatus. Of the seven cases with marked atrophy of the organ of Corti, in two the loss of hearing was considerably greater than the average. In these cases there was bilateral loss of hearing present. In the four cases of unilateral atrophy the audiograms were considerably lower than the average, but the loss on the atrophic side was greater in only two of them. In the other two cases of unilateral atrophy the audiograms for both ears were about the same. In the remaining one case the audiogram was about the average for the age.

Definite atrophy of greater or lesser degree was present in the spiral ganglion and nerve fibres of the lower basal coil in 17 cases (65.4 per cent). The number of the nerve fibres in the osseous spiral lamina varied from a moderate decrease to total absence in portions of the region. In all of these cases there were corresponding changes in the number of ganglion cells present. The loss of hearing was more than the average except in three cases. In these cases the loss of acuity of hearing for high tones and the decreased density of innervation of the lower basal turn appear to be correlated in a manner similar to those reported by Guild, Crowe, Bunch and Polvogt (1931).

Atrophy of the stria vascularis in the lower basal turn was observed in seven cases (27 per cent); in four of them it was unilateral. Appearances of early atrophy were seen in two cases. In five of the atrophic cases the loss of hearing was more than the average.

Atrophy of the cells of the external sulcus was observed in seven cases; in five of these there was also atrophy of the organ of Corti. The loss of hearing was more than the average in six of them.

Changes typical of the advanced arteriosclerosis of the larger blood vessels were not found in the small arteries of the meatus and modiolus. The alterations consisted rather of proliferation of the cells of the intima and subendothelial tissue, hypertrophy of the internal elastic lamina, thickening of the media, indistinctness of the outlines of the muscle cells. These resemble those seen in the small vessels of skeletal muscle by Anderson, Keith and Kernohan (1931).

Such changes were found in some of the meatus vessels in 15 cases (57.7 per cent); the loss of hearing was considerably greater than the average in 11 of them. In most of these cases with definite meatal vascular changes there was hypertension. Thus the condition of the meatus vessels would appear to be related to the loss of hearing and to the condition of the cochlear ganglion. However, this cannot be considered as a rule of absolute value because of the several exceptions.

The modiolar vessels are much smaller than the meatus vessels, the largest being almost of precapillary arteriole size. None were found with alterations which could be definitely stated to be arteriosclerotic in character. There was no appreciable difference to be found between the condition of the blood vessels of the lower basal turn and the other turns.

The question arises, what is the primary factor responsible for all these changes? The majority of the authors (Wittmaack, Bruehl,

Manasse, Stein, et al.*) are of the opinion that it is the arteriosclerosis. Some of the observations here reported seem to be in agreement with this opinion. In other cases, however, this explanation does not appear to be sufficient to explain the conditions found. Further investigation is obviously necessary.

While the typical loss of hearing, as has been shown above, is not constantly present, yet its presence may be regarded as an additional clinical sign indicating probable cerebral arteriosclerosis. Naturally the establishment of the diagnosis must be aided by the other symptoms of early cerebral arteriosclerosis.

LITERATURE.

BUNCH, C. C.: Age Variations in Auditory Acuity. Arch. of Otolaryngology, Vol. 9, p. 625, 1929.

BUNCH, C. C.: Further Observations in Auditory Acuity. Arch of Otolaryngology, Vol. 13, p. 170, 1931.

BUNCH, C. C., and RAIFORD, T. S.: Race and Sex Variations in Auditory Acuity. Arch. of Otolaryngology, Vol. 13, p. 423, 1931.

GUILD, S. R.; CROWE, S. J.; BUNCH, C. C., and POLVOGT, L. M.: Correlations of Differences in the Density of Innervation of the Organ of Corti, etc. Acta Oto-laryngologica, Vol. XV, Fasc. 2,4, p. 269, 1931.

KERNOHAN, ANDERSON and KEITH: The Arterioles in Cases of Hypertension. Arch. of Internal Medicine, Vol. 44, 1929.

STEIN, C.: Die Arteriosklerose des Gehoerorganes. Alexander-Marburg: Handbuch der Neurol. des Ohres, Vol. 21, p. 641, 1928.

ZWAARDEMAKER: Ueber das presbyakustische Gesetz an der unteren Grenze unseres Gehoers. Arch. für Ohrenheilkunde, Vol. XXXV, p. 299, 1893.

*Cited after Lederer: Die Altersveraenderungen im Gehoerorgan. Denker-Kahler Handbuch d. Hals-Nasen-Ohrenheilk., Vol I, 705.

ACUTE RETROPHARYNGEAL ABSCESS; REPORT OF CASES.*

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"The first notice of abscess of the pharyngoesophageal region dates as far back as the second century of the Christian era. Galen[1] reported a case in his own experience which terminated in spontaneous rupture."

Gillette[2], in 1867, demonstrated the lymph nodes and vessels in the loose connective tissue of the retropharyngeal space.

John Bokai[3], in 1876, showed that the majority of these abscesses resulted from retropharyngeal lymphadenitis independent of tuberculous caries of the vertebrae. More recently Berg[4], Mosher[5], Frank[6], Glogau[7], Wishart[8], Douglas Guthrie[9] and H. P. Chatellier[10] have added to our knowledge of retropharyngeal abscess.

Etiology: Acute retropharyngeal abscess of lymphatic origin occurs more frequently in childhood, three months to three years; 75 per cent occur in the first three years of life[11]. Undernourished children and those debilitated from serious illnesses are prone to suppurative inflammation of the connective tissue of the nose and pharynx. Hence retropharyngeal abscesses occur in children following: *1.* Sinusitis; *2.* adenoiditis; *3.* tonsillitis; *4.* angina associated with influenza, measles, scarlet fever and diphtheria; *5.* otitis media; *6.* trauma incident to removal of tonsils and adenoids or that caused by the impaction of foreign bodies in the nasopharynx, as metal buttons and fish bones.

Acute retropharyngeal abscess is observed in adults as a complication of: *1.* Posterior sinus disease; *2.* otitis media or mastoiditis; *3.* carious teeth; *4.* trauma from *a.* blows, *b.* swallowing hard substances, and *c.* incident to tonsillectomy.

The exciting micro-organism is most frequently the streptococcus, less often the staphylococcus, pneumococcus or Klebs-Loeffler bacillus.

Anatomy: In opening a retropharyngeal abscess through the mouth the tissues passed through from before backward are: *1.* Pharyngeal mucosa; *2.* pharyngeal aponeurosis; *3.* superior constrictor muscle; *4.* bucco pharyngeal fascia; *5.* into the loose alveolar tissue of the retropharyngeal space[8]. These structures constitute the anterior, and the prevertebral fascia the posterior, boundary of this space. In the midline the connective tissue is condensed into a

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fairly consistent raphe, which binds the pharyngeal mucosa to the spine, so that suppuration in the space on one side does not readily spread to the other side. Laterally it is separated only by a thin lamella, the ala fascia, from the parapharyngeal[12] space[7], sometimes designated as the pharyngomaxillary fossa. Vertically the retropharyngeal space extends from the base of the skull above to the farthest prolongation of the cervical fascia below. The retropharyn-

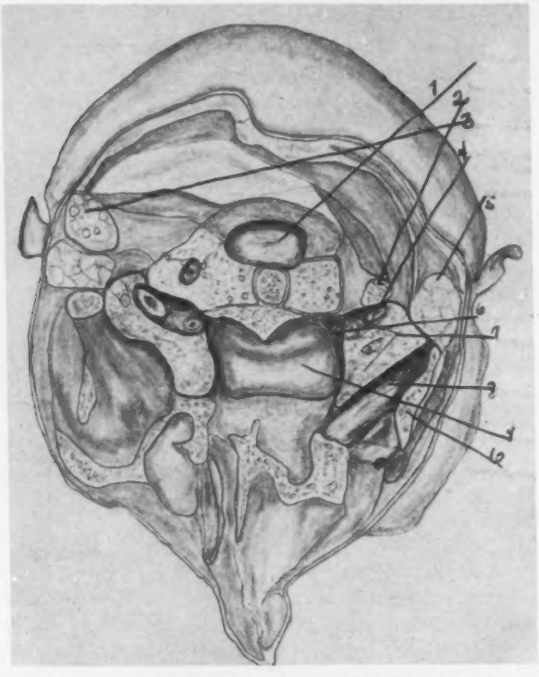

Plate 1. Cross-section at level first cervical vertebra showing pharyngomaxillary fossa. 1. Foramen magnum. 2. Neck of mandible. 3. Mastoid Process. 4. Carotid Sheath with Vein, Artery and Nerve. 5. Parotid Gland. 6. Superior Constrictor. 7. Pharyngo-Maxillary Fossa. 8. Internal Pterygoid. 9. Pharynx. 10. Ramus of Mandible.

geal lymph glands lie in this space. They vary in size and number, 8 to 10 in infancy. After three to eight years of age they atrophy, so that in adults there remain, as a rule, only one or two glands on each side. These are the ones which suppurate in retropharyngeal abscess.

The pharyngomaxillary fossa[5] is a funnel-shaped space. Its base is the base of the skull and its point is opposite the lower limit of

the angle of the jaw. The carotid sheath emerges at the point of the funnel and for practical purposes continues the fossa through the neck to the thorax. The internal boundary of the fossa is the superior constrictor muscle, the external is the inner surface of the ascending ramus of the mandible, covered by the internal pterygoid muscle. Superiorly the inner prolongation of the parotid gland makes a part of the external boundary. The upper cervical vertebrae, covered by the prevertebral muscles and fascia, make the posterior. The fossa is divided by the styloid process and the muscles which spring from it into a large anterior chamber, which is in relation to

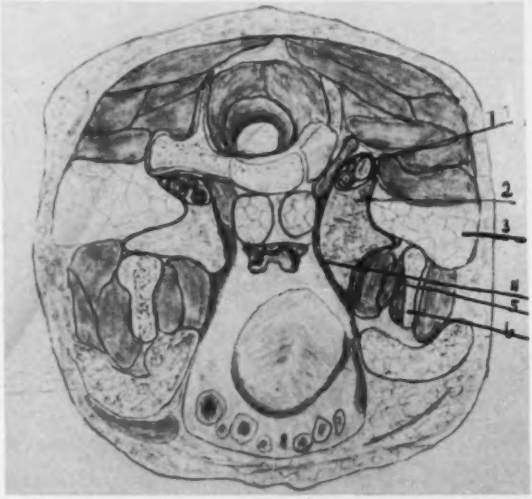

Plate 2. Pharyngo-maxillary fossa at birth. 1. Carotid Sheath. 2. Fat in Pharyngo-Maxillary Fossa. 3. Parotid Gland. 4. Superior Constrictor Muscle. 5. Internal Pterygoid Muscle. 6. Ramus of Mandible.

the tonsil; and a smaller posterior chamber containing in its outer part the internal carotid artery, the jugular vein and the pneumogastric and sympathetic nerves, medially and separated only by a thin lamella of fascia is the retropharyngeal space just described (see Figs. 1 and 2). From these relations it will be seen that retropharyngeal suppuration, left to itself, may: *1.* Burst spontaneously and drain itself into the pharynx. *2.* Travel downward behind the esophagus into the posterior mediastinum. *3.* Burrow laterally into the pharyngomaxillary fossa, behind the great vessels and the sternocleidomastoid muscle, and bursting through the fascia point in the anterior or posterior triangle of the neck. *4.* The pus may be guided

downward by the prevertebral fascia to the lower part of the neck, thence along the sheath of the axillary vessels or beneath the clavicle.

Pathologically: There is first a sinusitis, an adenoiditis, tonsillitis or middle ear infection which spreads along the lymphatics, producing an adenitis of the retropharyngeal lymph glands (this stage of induration and swelling may be overlooked), then periadenitis, and finally abscess formation.

The diagnosis is based upon: *1.* A history of an upper respiratory infection. *2.* Dysphagia, which in addition to being painful becomes mechanical. The collection of pus accumulates and of its weight spreads toward the bottom of the pharynx. It results in progressive

Fig 229. Retropharyngeal abscess. Section through the cervical vertebrae, pharynx and adjacent parts of a boy, age 2½ years, showing the cavity of a retropharyngeal abscess. The abscess extends from the level of the basi-occipital above, to that of the fifth cervical vertebra below; the swelling is greatest opposite the body of the third cervical vertebra. The abscess cavity is lined by a brownish membrane; the posterior pharyngeal wall, which forms the anterior boundary of the cavity, appears thickened. The way in which pressure has been exercised on the upper opening of the larynx is well illustrated. There is no evidence of spinal caries, nor anything to explain the cause of the abscess. (St. Bartholomew's Hosp. Mus., No. 1841a).

inanition from day to day. *3. Dyspnea:* Inspiration is especially difficult. Pressure forward of the low lying protrusion encroaches on the larynx and produces a mechanical dyspnea (see Fig. 229. From Sir St. Clair Thomson: Diseases of Nose and Throat). In certain severe cases spasms of the glottis complicate the prognosis, while in still others irritation of the peripheral filaments of the vagus accounts for some of the respiratory difficulty. *4.* There may be cervical adenitis on one or both sides. *5.* Croupy cough, vomiting

and reflux of fluids through the nose are symptoms of secondary importance. *6.* On inspection and palpation a fluctuating paramedian protrusion of the posterior pharyngeal wall is found. Fluctuation may not always be demonstrable. Plunging an aspirating needle into the pharyngeal swelling may enable one to make sure of the presence of pus.

A number of conditions should be differentiated, as adenoiditis, croup, laryngeal diphtheria, edema of the glottis, foreign body in the larynx, aneurysm[6] of the carotid, malignancy[13], osteomyelitis of the spine, cervical scoliosis, tuberculosis of the cervical spine.

Complications of retropharyngeal abscess are of several types: *1.* Incident to the effect on the blood vessels locally there may take place thrombosis or erosion of the vessels, with resultant hemorrhage. *2.* The infection may spread by way of the lymphatics or blood stream, producing septicemia or pyemia. *3.* Any one of a group of conditions may arise as a result of mechanical embarrassment to deglutition, or respiration, or following spontaneous rupture of the abscess; as inanition, asphyxia from mechanical or spasmodic laryngeal stenosis, suffocation from aspiration of pus and detritus, or pneumonia. I have been able to verify the following complications from the literature, the experience of my colleagues and myself.

1. Hemorrhage: In one of my cases there was hemorrhage from the abscess cavity on two different occasions. Pearson[14], Travers[15], and Liang[16] had cases of secondary hemorrhage in which the common carotid was ligated, with recovery. Lidell[17] reported a case of hemorrhage from the facial, where the ligatures cut out and the hemorrhage was finally stopped with actual cautery. In another case, one of hemorrhage from the superior thyroid, the ligatures cut out and the bleeding was finally controlled with lunar caustic.

Cases of fatal hemorrhage are reported by Lidell, Wylie and Wingrave[18], Wishart[8] and Savory[19].

2. Thrombosis of the internal jugular[6, 7, 12].

3. Edema of the larynx.

4. Laryngeal stenosis from pressure, requiring tracheotomy[20, 21].

5. Asphyxiation from spontaneous rupture[22].

6. Lung abscess was present in Dr. Wegner's Case 3.

7. Paralysis of the esophagus[23].

8. Septicemia, Dr. Wiedman's Case 7.

Treatment: During the prodromal stage and that of adenitis and periadenitis hot, moist compresses should be applied to the neck and renewed frequently. In addition steam inhalations, hot gargles, supportive measures of feeding, elimination and even vaccinotherapy

may prove helpful. When the stage of suppuration is reached the one curative treatment is operation. No general anesthetic is given. Children are restrained by being securely swathed in a sheet or blanket to prevent movement of the arms and legs. The patient is placed on his back or side with the head low. No mouth gag is used. The mouth is held open by tongue depressor or palpating finger. A scalpel, guarded to one-half-inch of the point with adhesive tape, is plunged into the pharyngeal swelling and carried upward to make an ample incision, or a sharp-pointed hemostat or tonsil scissors may be pushed into the walls of the abscess and the points spread apart while being withdrawn. The patient is immediately turned on his face to avoid aspiration of pus into the trachea. Suction and expression may be used to complete the evacuation of the abscess.

When the infection extends along the fascial planes to the pharyngomaxillary fossa or any of the deep cervical lymphatic glands, particularly the posterior carotid chain, external incision is indicated. Whether incision is made anterior or posterior to the sternocleidomastoid varies with the location of the pus in the individual case.

In attacking the abscess by the anterior[24] route a vertical incision is made in the infrahyoid region through skin, superficial fascia, platysma and superficial layer of the deep fascia to the anterior border of the sternocleidomastoid. The index finger is now passed between the vascular fascia and the visceral fascia down to and through the thin lamella, the ala fascia which connects the two. The finger is now in the retropharyngeal space. The abscess now usually presents as a tense bag which can be opened with hemostat or other short, sharp instrument.

More recently I have been able to reach and drain satisfactorily the pharyngomaxillary fossa by the submaxillary approach as described by Mosher[26]; that is, adequate incision over the submaxillary gland. After exposing the gland it is turned upward and forward out of its bed. One is then able to pass a finger upward between the ascending ramus of the mandible and the carotid sheath and feel the end of the styloid process. The finger is then in the pharyngomaxillary fossa and one is able to reach pus in either the anterior compartment, with which the tonsil is closely associated, or in the posterior compartment, in which lies the carotid sheath.

CASE REPORTS.

Case 1: R. G., a young woman, age 20 years, was seen because of recurrent sore throat and persistent albuminuria.

Tonsillectomy was performed April 6, 1929, under local infiltration anesthesia. On the third day after operation she complained of pain

in the left posterior cervical region. Her temperature was 100°, pulse 100, respiration 18. By the fifth day postoperative she complained bitterly of pain in the left side of the neck, more than the local findings seemed to warrant. There was some swelling of the cervical glands of the left side in both anterior and posterior triangles. Her temperature was 102.4°, pulse 120, respiration 18. She was sent to the hospital. There hot saline gargles, copious hot boric dressings to the left cervical region and supportive treatment were instituted. She received during the progress of the case two deep Roentgen therapy treatments and three applications of the violet ray.

Fig. 3. Patient subject Case Report 1. "A" and "A'" incision April 16, 1929. From there no pus was obtained. "B" site of infraclavicular incision, April 20. From this pus drained freely for 12 days. "C" incision posterior to sternomastoid, May 8. Drainage, 18 days.

The swelling of the tissues of the left side of the neck and the area of redness just below and posterior to the auricle increased but there was no fluctuation. The tenth day after tonsillectomy the patient lay with head back, mouth open and saliva drivelling, rather than endure the pain of swallowing. When she attempted to swallow liquids part of them regurgitated through the nose. There was marked swelling of the lateral wall of the pharynx high up and behind the posterior pillar. From this free pus discharged. Under gas anesthesia incision was made both in front and behind the sternocleidomastoid muscle and carotid sheath, blunt dissection carried

deeply into the neck and drainage tubes inserted. No macroscopic pus was obtained at that time or later.

Pus continued to discharge freely from the left retropharyngeal space; two days later, following a fit of coughing, there occurred a vigorous hemorrhage from the abscess cavity. On mopping away the clots the opening of the abscess cavity seemed to be plugged with a clot and free bleeding had ceased. Forty-eight hours later another lesser hemorrhage occurred. On the morning of the fourteenth post-operative day fluctuant swelling was discovered in the left infra-clavicular region. This was incised and an ounce of foul, grumous pus evacuated. Culture of the pus showed fusiform bacilli and staphylococci. Drainage from both the retropharyngeal and infra-

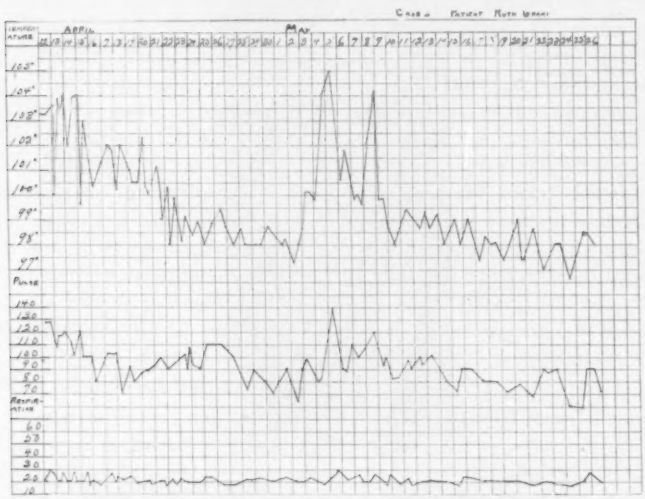

Fig. 4. Temperature, pulse and respiration Case 1.

clavicular space gradually diminished and after nine days normal temperature patient was discharged from the hospital. The following day she was readmitted. Her temperature was 102° and she complained of pain in the left posterior cervical region, high up near the occiput. The white cell count was 13,800. After six days a fluctuant swelling had localized low in the left posterior cervical triangle. This was incised under novocain anesthesia and an ounce of creamy pus obtained (see Fig. 3). Subsequent convalescence was uneventful and patient was discharged recovered on the fiftieth day following her tonsillectomy (see Fig. 4).

In October, 1929, patient was pursuing her studies in apparently good health, but her urine still continued to show a trace of albumin.

Case 2: Baby L. W., age 8 months, was seen by me, Dec. 6, 1920, because of difficulty in breathing and swallowing. The child had had an upper respiratory infection for 10 days. One week before, because of difficult breathing, a physician had attempted to remove the baby's adenoids with his finger. On examination temperature was 102° R., respiration was embarrassed. There was difficulty in swallowing and a bulging, fluctuating mass on the left side of the postpharyngeal wall could be seen and palpated. With the child securely swathed in a blanket and held firmly against the shoulder of the father the abscess was incised with a guarded scalpel and divulsed with a hemostat. The child was immediately turned on its face and considerable thick, creamy pus was drained from the abscess. The following day temperature was normal and the postpharyngeal swelling had almost completely disappeared.

Case 3 (reported through the courtesy of Dr. E. S. Wegner): The patient, an infant, age 6 months, was admitted to the hospital March 6, 1927, because of high temperature (107° the night before) and difficult breathing. The baby had had a pneumonia and marked swelling of the glands of the neck; for two weeks the temperature had fluctuated from subnormal to 103.5° and 107°. She had had a good appetite throughout her illness. Took food and water eagerly, but choked easily and frequently, and swallowed with more and more difficulty.

On examination the first thing noted is stridor. Baby lies with head thrown back, eyes almost bulging and mouth wide open. Breathing is very difficult and noisy. The chest presents a peculiar bulging at the lower end of the sternum. This looks and feels like the thickening of the sternum and the rib border. On the right side of the neck and below and anterior to the angle of the jaw is a swelling, which seems to extend down into the chest and apparently is attached to the deep structure of the neck. The swelling is the size of a lemon and feels spongy. The mucous membrane of the mouth and throat are dry and red, the whole postpharyngeal wall bulges, and on palpation is soft and fluctuating. Temperature, 104.6°; pulse, 175; respiration, 54-68; W. B. C., 32,600. Roentgenogram showed a postpharyngeal mass extending from the occiput to the level of the second dorsal vertebrae bulging anteriorly, most markedly in the region of the hypopharynx.

Diagnosis: 1. Mediastinal tumor? 2. Pus in the mediastinum, later changed to postpharyngeal abscess, burrowing into mediastinum (see Fig. 5).

The abscess was incised from within the mouth and the opening in the abscess wall repeatedly divulsed. Ten days later rib resection was done for drainage of a lung abscess. At the time of the rib resection no pus was obtained, although the Roentgenogram showed a shadow on the right side, extending from the second to the sixth rib. Three days later the abscess discharged spontaneously and closed at the end of five or six days. Child was discharged from the hos-

Fig. 5. Lateral Roentgenogram, Case 3. Shows boundary of retropharyngeal abscess, occiput to second thoracic vertebra.

pital 61 days after admission. Subsequent health history has been uneventful.

Case 4: Baby R. R. A., age 8 months, was seen with Dr. E. V. Wiedman, Jan. 8, 1930. Jan. 1, the baby developed temperature and what appeared to be a beginning bronchopneumonia. At the end of the fourth day there was a swelling at the angle of the jaw on the right side of the neck. By Jan. 7 the swelling had become markedly larger, breathing was very difficult and he was unable to nurse on

account of the difficulty in swallowing. The baby was pallid and dehydrated. Temperature, 103.4°. Inspiration made with great effort. The mouth was difficult to open; when forced open with a tongue blade he almost stopped breathing. On inspection and palpation of the pharynx, a bulging, fluctuating mass, posterior to the right tonsil and extending to the midline, was found. The mass at the angle of the jaw on the right side was soft, but did not definitely fluctuate.

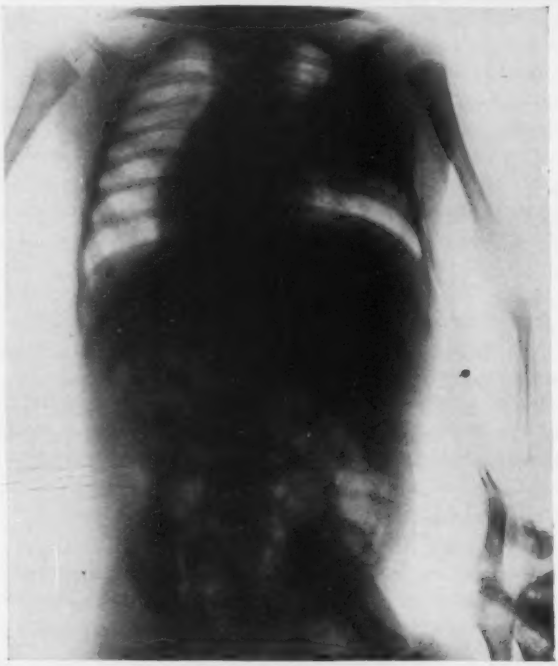

Fig. 6. Roentgenogram, anterior posterior, Case 3. Shows abscess, right lung, viewed from behind, second to sixth rib.

With sandbag under the shoulders and head low, the abscess was opened by plunging a rather sharp-pointed hemostat into the most prominent point of the pharyngeal swelling. The hemostat was guided by the palpating finger. The child was immediately turned over on his face. An ounce of green, watery pus was expressed and sucked out of the throat.

Twice during the convalescence the temperature rose to 101° or 102°. Examination showed that the opening in the pharyngeal wall

had become plugged. This was divulsed and drainage re-established.

One-half of an erythema dose of Roentgen ray was given the glands at the angle of the jaw on the right side of the neck.

The patient was discharged from the hospital at the end of four weeks.

Case 5: Baby R. L., male, age 1 year, had had an upper respiratory infection up and down for six or eight weeks. There had been tonsillitis, both ears had discharged, temperature at times. He was fussy, irritable, sleep was restless, did not eat well, and the parents feared mastoid disease.

On examination the baby was pale and looked sick. Muscles were soft and flabby. The right ear was dry. The left showed some dry detritus at the bottom of the canal. The throat showed a fluctuant swelling in the right posterior pharyngeal region, low down. In the right cervical region at the angle of the jaw there was a diffuse mass, which seemed to fluctuate. There was a marked excoriation of the buttocks. Diapers smelled strongly of ammonia. Temperature, 102°. Blood examination showed: Hemoglobin, 52 per cent; erythrocytes, 3,700,000; leukocytes, 16,500. A differential count showed: Polynuclears, 46 per cent; large lymphocytes, 26 per cent; small lymphocytes, 22 per cent, and transitional forms, 6 per cent.

Diagnosis: Retropharyngeal abscess, right, acute.

Treatment: Incision at the right of the median line on the level of the hypopharynx with a sharp-pointed tonsil scissors yielded half an ounce of greenish, yellow pus. Subsequently the diet was supervised, cod liver oil and iron were administered and one-third of an erythema dose of Roentgen ray given the cervical glands. Discharged at the end of a week.

Case 6: Baby S., male, age 6 months, was seen at the request of Dr. Hans B. Mueller because of difficult breathing. The baby had been sick two weeks with tonsillitis, follicular, acute. For several days there had been swelling of the glands at the angle of the jaw, more marked on the left side. The last 48 hours breathing had become progressively more difficult and he refused to nurse or take the bottle.

On examination the baby was pale to blue. He lay with the head back. Breathing was stertorous. The tonsils were swollen and red. A fluctuant swelling of the left posterior pharyngeal wall, low down, bulged out like a walnut.

As the baby was in extremis we at once placed him on his side with the head low and opened the abscess with a sharp-pointed hemostat, guided along the palpating finger. Fully an ounce of pus was

expressed. This on culture showed staphylococci predominating. At the end of two days the patient was much improved and made an uneventful recovery.

Case 7: S. K., girl, age 3 years, was a twin, premature; birth weight, six pounds. She had measles, March 7, 1930. Apparently recovered, was normal for about three days, then began to run a temperature. About this time parents were having what was diagnosed as influenza. The mother had a peritonsillar abscess incised. The patient had a tonsillitis, and myringotomy was performed on both ears. By March 26 the dysphagia had been marked for two or three days. The mother stated that the child had nearly choked to death several times, but had been better in that regard that day.

On examination, March 26, there was some mucopurulent discharge in meatus, right ear. The discharge from the left ear was more profuse, watery and bloody in character. There was bilateral cervical adenitis and some swelling of the soft tissues of the face on the right side. Pus was discharging from the nasopharynx. No definite bulging or fluctuant mass was present, as I think the abscess had ruptured. One could wipe away the pus and it immediately recurred, apparently on the right side. The abscess drained freely for about three days. March 31, the temperature continued and there was free bleeding from the left ear, nose and throat. Tampon of both anterior and posterior nares failed to check the bleeding completely. The heart and lungs showed no evident lesion. Abdomen was tympanitic. There were a few purpuric spots, one-half to one c.m. in size, on lower abdomen, and mesial aspect upper part of the thighs. The urine showed albumin, casts and pus; and the blood: hemoglobin, 42 per cent; red cells, 3,380,000; leukocytes, 26,000. Schilling count: Metamyelocytes, 18 per cent; band forms, 49 per cent; segmented forms, 25 per cent; monocytes, 2 per cent; lymphocytes, 6 per cent. Coagulation time, 2 minutes; bleeding time, 7½ minutes. The micro-organism was not isolated, but it was thought we had to deal with a strain of the streptococcus hemolyticus.

One hundred and ten c.c. of whole blood were given intramuscularly. April 1, 75 c.c. of the father's whole blood were given into the vein. The purpuric spots increased in number and the patient died the evening of April 1.

Postmortem: Permission was not given to open the head, but interrogation of the nasopharynx shortly after death revealed on the right posterior pharyngeal wall a sloughed area, eight to 10 m.m. in diameter, from which a thin, watery discharge escaped. This was the point from which we had observed the pus to come on March 26.

The same small hemorrhagic spots were found in the small intestine, pericardium, pleurae, lungs and mucous membrane of the gall bladder.

The summary of anatomical lesions was bilateral fibrinous, purulent pleuritis, bronchopneumonia left, perihepatic and perisplenic adenopathy, myocarditis, endocarditis with vegetations right auricular and ventricular dilatation. Probable cause of death: septicemia, probably hemolytic streptococci.

Case 8: D. C., a boy, age 11 years (a patient of Dr. E. V. Wiedman and Dr. Stanley Zemer, with whose permission I report this case). Feb. 20, the patient began running a temperature, thought to be due to influenza. March 12, he complained of dysphagia, earache, pain in the right side of the neck and poor appetite.

On examination the boy is rather large for his age, has lost some weight and is pale. There is considerable purulent nasal secretion. The tonsils are out thoroughly. The cervical glands on the right are swollen, hard and painful.

On March 19 the right posterior pharyngeal wall presented a fluctuant swelling and needling of the affected cervical glands showed thick, creamy pus. Temperature, 104.4°. The abscess was incised through the posterior pharyngeal wall and also external incision was made anterior to the sternocleidomastoid down to the suppurating glands and drainage inserted. Culture of pus from the glands showed staphylococci predominating, a few short-chain streptococci and a few encapsulated diplococci. The retropharyngeal abscess drained and in a few days the pharynx was practically normal, but the neck did not heal. Temperature fluctuated and in spite of heat and Roentgen therapy locally there developed fluctuant swelling in the right posterior cervical triangle of the neck, low down. April 10, incision was made posterior to the sternocleidomastoid and drains inserted. This pus showed short-chain streptococci. Discharge from this incision gradually diminished and the patient was discharged, April 27, 1930.

COMMENTS.

Case 1: I was interested to know why the pus did not descend into the mediastinum and cause death. I believe that the pus was guided down by the superficial layer of the deep fascia and the pretracheal fascia between the clavicle and the first rib into the infraclavicular space. In the posterior triangle of the neck the pus must have been confined between the prevertebral fascia posteriorly and the carotid sheath anteriorly and the superficial layer of the deep fascia externally.

I felt that in carrying out the infiltration anesthesia incident to the tonsillectomy I had possibly passed the needle through infected tonsil tissue and the constrictor muscle of the pharynx into the pharyngomaxillary fossa, thus infecting it. I have since that experience tried to follow as nearly as possible the block anesthesia of Smith[25].

Cases 2, 4, 5, and 6 are rather typical of the acute retropharyngeal abscess of lymphatic origin. They emphasize the necessity of early recognition and prompt evacuation of the abscess.

Case 3 was interesting because of the apparent extension of the pus into the mediastinum and complicating lung abscess with recovery.

Case 7 illustrates the second major type of complication of retropharyngeal abscess, namely, septicemia or pyemia.

Case 8: The occurrence of retropharyngeal abscess, infection of the pharyngomaxillary fossa and glands in relation to the carotid sheath below the apex of the fossa in a patient whose tonsils had been removed previously was unusual.

CONCLUSIONS.

1. Retropharyngeal abscess following tonsillectomy under local anesthesia constitutes one of the objections to performing the operation under this type of anesthesia. Its incidence can be reduced, if not prevented, by careful block anesthesia.

2. Retropharyngeal abscess is more common than is commonly supposed. It is often overlooked. Careful examination of the pharynx should be made in all infants presenting any one or all of the following: *a.* Upper respiratory infection. *b.* Dysphagia. *c.* Dyspnea and cervical adenitis.

3. When recognized early and treated promptly, the prognosis is good.

4. In abscesses involving the pharyngomaxillary fossa and carotid sheath, adequate incision anterior to the sternomastoid will most often be rewarded by reaching and evacuating the abscess.

BIBLIOGRAPHY.

1. MACKENZIE, MORRELL: Manual of Diseases of Nose and Throat, 2:57. Quoted by Goldstein. See reference No. 20.
2. GILLETTE, E. P.: Des Abces. Retropharyngiens Idiopathiques, Paris, 1867.
3. BOKAI, JOHN: Ueber Retropharyngeal Abscesse bei Kindern. Jahrs f. Kinderch, 10:108, Leipsic, 1876.
4. BERG, H. W.: Causation, Pathology and Symptoms of Retropharyngeal Abscess. Med. Rec., 45:522, April 28, 1894.
5. MOSHER, HARRIS P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular Vein. THE LARYNGOSCOPE, 30:365, June, 1920.

6. FRANK, IRA: Retropharyngeal Abscess. Tr. Section Laryngol., Otol. and Rhinol., A. M. A., 184, 1921.

7. GLOGAU, OTTO: Abscesses Descending from Upper Air Passages Into the Neck and Mediastinum; Typical External Operation with Prophylactic Mediastinotomy. Tr. Amer. Acad. Ophthalmol. and Oto-Laryngol., 393, 1922.

8. WISHART, D. E. S.: Retropharyngeal Abscess: Erosion of Internal Carotid with Pathological Specimen. *The Canadian Med. Assn. Jour.*, 13:635, Sept., 1923.

9. GUTHRIE, DOUGLAS: Acute Retropharyngeal Abscess in Childhood. *British Med. Jour.*, 2:1174, Dec., 1925.

10. CHATELLIER, H. P.: Diagnostic et Traitement des Abscess Retropharyngien. Medicine, Paris, 1926. VII:299-301.

11. THOMSON, SIR ST. CLAIR: Diseases of the Nose and Throat. D. Appleton & Co., New York, 1927.

12. WALDAPFEL, RICHARD: Post-Tonsillitis Pyemia. Tr. Amer. Acad. of Ophthalmol. and Oto-Laryngol., 291, 1928.

13. DOWNIE, W.: Retropharyngeal Abscess. *Glasgow Med. Jour.*, March, 1903.

14. PEARSON, JOHN: Principles of Surgery. London, 99, 1788.

15. TRAVERS, FRED I.: Retropharyngeal Abscess, Secondary Hemorrhage, Ligation of Internal Jugular and Common Carotid on Left Side, Recovery. *British Med. Jour.*, 2:703, 1902.

16. LIANG, P. R.; LEI, C. H., and LIANG, P. T.: Retropharyngeal Abscess, Secondary Hemorrhage, Ligature Common Carotid, Recovery. *British Med. Jour.*, 967, May 23, 1925.

17. LIDELL, J. A.: Abscesses of the Neck. *Amer. Jour. Med. Sci.*, 85-86: 321, 1883.

18. WYLIE, A., and WINGRAVE, V. H. W.: A Case of Retropharyngeal Abscess in a Female Aged 21 Years. Evacuation of Pus for Dyspnea. Death from Sloughing of the Internal Carotid. Lancet, 1:1042, April 14, 1906.

19. SAVORY: Quoted by Treves, Frederick, Applied Anatomy, Philadelphia, Lea Brothers & Co., 144, 1901.

20. GOLDSTEIN, MAX A.: Retropharyngeal Abscess: Report of Some Unusual Cases. THE LARYNGOSCOPE, 18:46, July, 1908.

21. CLINE, L. C.: Retropharyngeal Abscess. THE LARYNGOSCOPE, 18:668, September, 1903.

22. CHARLTON, C. COULTER: Retropharyngeal Abscess with Rupture, Asphyxiation and Death Following an Acute Attack of Tonsillitis. THE LARYNGOSCOPE, 24:985, December, 1914.

23. THOMPSON, JOHN A.: Retropharyngeal Abscess with Paralysis of Esophagus. THE LARYNGOSCOPE, 21:1081, November, 1911.

24. DEAN, L. W.: The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebrae. Ann. Otol., Rhinol. and Laryngol., 28:566, June, 1919.

25. SMITH, ARTHUR R.: Block Anesthesia and Allied Subjects. St. Louis, C. V. Mosby Co., 769, 1920.

26. MOSHER, HARRIS P.: The Submaxillary Approach to Deep Pus in the Neck. Tr. Amer. Acad, Opthalmol. and Oto-Laryngol., 35, 1929.

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OBSERVATIONS ON PARALYSIS OF THE RECURRENT LARYNGEAL NERVE.*

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Loss of motion or paralysis of one vocal cord, partial or complete, due to a lesion of the recurrent laryngeal nerve is a common occurrence. It is curious that this subject appears to receive so little attention. I do not recall to have seen over a long period any paper listed on the programs of this Section, nor have I heard any case reports of this disorder, nor have there been any patients presented here with the same. Possibly I am bringing up a subject which to the experienced laryngologist is an every day experience, but I am rather certain that the younger men in this practice are not so familiar with it. I would invite the interest of the latter group since the detection of this lesion and the attempt to track down its cause may become an absorbing adventure in the wider realms of internal medicine and general surgery. It thus presents another opportunity of escape from narrowing and merely mechanical specialism (*i. e.*, just surgical skill), an escape so greatly needed by the developing rhinolaryngologist.

I will not burden you with a rehearsal of the physiology and anatomy of the larynx, nor detail the causes known to be responsible for lesions of the recurrent laryngeal nerves. These facts, I am sure, are known to all and are, moreover, fully described in the textbooks. I wish only to present several viewpoints based entirely on clinical observation, which may serve as a stimulus to those to whom this approach to the subject will seem novel.

It is probably well known by all that the anatomical arrangement of the recurrent laryngeal nerves is in a great measure responsible for lesions of the same. Nature seems to have erred when she planned the innervation of the larynx. Instead of carrying the nerves directly to their terminus, encased and supported by surrounding structures and, so to speak, insulated and protected by them, she has allowed these two vitally important nerves to dangle from the main trunk line, thus subjecting them to numerous hazards. On the basis of structural arrangement we should then expect that the longer and more exposed left nerve will suffer damage more frequently than

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the shorter right, and this inference is fully borne out by the clinical evidence.

I may be taken to task for claiming that paralysis of the recurrent nerve is a frequently occurring lesion, but clinical evidence favors the opinion. I am under the impression that loss of motion of the vocal cord, due to a lesion of the recurrent nerve, is frequently overlooked. Recognition of this lesion in its terminal stage, with the cord in cadaveric position, or with distortion of the larynx, or with muscle atrophy, or inability of the sound cord to effect closure of glottis—all these appearances will help to establish a diagnosis. It is the earlier stages of the lesion that appear to escape observation. This failure to detect the less striking examples of loss of motion of the vocal cord is due for the most part to a definite optical illusion during examination. The observer who looks fixedly at the vocal cords during phonation will appear to see an excursion of both toward the median line, whether both are functioning normally or whether only one performs. It is, of course, well understood that the sound cord usually swings beyond the median line in its effort to effect the closure of the glottis, and it is this overaction that gives the appearance of motion to a really motionless cord. The tyro will therefore be deceived.

There is, however, a technique of examination which will readily disclose any loss of motion of a vocal cord, whether slight or marked. This method is not to my knowledge mentioned in the textbooks, nor does it seem to be widely understood. Briefly stated, we have found that the proper method for positive diagnosis lies in watching the arytenoids during phonation and not to concentrate on the cords during this function. It will then be seen unfailingly that the arytenoid of the affected side remains fixed, while the normal one moves against it or impinges upon it. Simultaneously, the cords appear to be moving against each other also. The latter is the optical illusion referred to. There cannot, of course, be any independent movement of a cord if its arytenoid fails to move. I will not discuss at length the significance of the slight twitch of the arytenoid of the affected side often noted during attempt of phonation. The reason for this phenomenon, which serves to differentiate the loss of motion under discussion from arytenoid ankylosis, is well understood.

We have stressed this method of examination with our students at the college and it is gratifying to note how readily they have been able to recognize any hypokinetic lesion of the vocal cords.

It is further easy to see why many cases of unilateral recurrent nerve palsy escape observation when it is stated that the greater num-

ber of patients in whom this disorder occurs do not originally present themselves for the relief of any disturbance of voice. The surgeon is likely, therefore, in crowded clinic practice to pass over the examination of the larynx or to inspect it hastily.

It is an ordinary experience to hear many patients describe their ailments of throat or nose or ear, who give no history of hoarseness and in whom undeniable recurrent nerve paralysis is discovered. Attention to the character of the voice naturally ensues upon this discovery and often nothing more than a lack of resonance is noted. Questioning occasionally elicits the statement that sustained vocal effort is difficult, especially in the case of singers or speakers; but the general run of individuals declare nothing to be wrong in the every day use of the voice.

A field of great extent and wide interest unfolds to the view of the laryngologist who unexpectedly encounters a loss of motion of a vocal cord. Obviously, he has first to minister to the ailments complained of, concealing however the fact of impairment of the vocal cord. The psychological effect of such a disclosure can only be harmful, the word paralysis having in the lay mind a sinister connotation. A less forbidding expression, such as loss of motion or weakness of the cord, would serve much better if after a general survey of the patient the information must be given.

The co-operation of the internist or general surgeon is to be enlisted when the laryngologist fails to discover a local cause, or one remote from the larynx. This may sound very trite, but I do not think that such a joining of effort occurs invariably. The general examination may disclose no cause or it may reveal some serious condition which had not yet given rise to symptoms. In other words, recurrent nerve involvement may be the first discovered indication of a disease remote from the larynx.

For the purpose of this paper I am presenting a series of cases of recurrent laryngeal nerve paralysis of one side, 20 in number. These cases were observed during a period of from six to eight weeks at the Vanderbilt Clinic. There were more observed than the number here reported, but to include them will only be a repetition in kind. In recounting to you briefly the history of these cases I have given the patient's own statement of his ailment, the diagnosis of the conditions found and also such data as will confirm the views offered in this paper.

Group 1: Persons with loss of motion of one cord without dysphonia.

Group 2: Persons with loss of motion of one cord, without dysphonia, but observed lesions elsewhere suggesting cause.

Group 3: Persons with loss of motion of one cord, markedly hoarse, and coming for relief from this condition.

Group 1: Patients with Loss of Motion of One Vocal Cord Without Impairment of Voice.

U. O., male, age 40 years. Noises in both ears for one year. *Diagnosis:* O. M. C. C. Deviated septum, tonsil stumps, small adenoid. *Collateral Finding:* Loss of motion L. vocal cord.

W. J. R., male, age 32 years. Sore throat. *Diagnosis:* Hypertrophic infected tonsils. *Collateral Finding:* Loss of motion L. vocal cord.

J. M., male, age 14 years. Nose feels full, snuffles and tries to clear throat. *Diagnosis:* T. and A., acute maxillary sinusitis. *Collateral Finding:* Loss of motion L. vocal cord.

L. D., male, age 25 years. Sore throat for six weeks, especially on right side. *Diagnosis:* Infected tonsil stumps, adenoid remains. *Collateral Finding:* Loss of motion L. vocal cord. Some hyperemia in larynx.

B. H., male, age 30 years. Ears feel blocked up. *Diagnosis:* Acute rhinitis, acute pharyngitis, subacute tubotympanitis. *Collateral Finding:* Loss of motion L. vocal cord, some thickening of both cords.

N. S., male, age 30 years. Intermittent attacks of headache, frontal, for two months. *Diagnosis:* Chronic pharyngitis, probable right frontal and maxillary sinusitis. *Collateral Finding:* Loss of motion L. vocal cord, moderate interarytenoid thickening.

F. B., male, age 45 years. Nasal obstruction, asthma. *Diagnosis:* Acute rhinitis and pharyngitis. Left maxillary sinusitis. *Collateral Finding:* Loss of motion L. cord.

J. E., male, age 34 years. Running nose, frequent colds, cough. *Diagnosis:* Deviated septum, subacute rhinitis, hypertrophied tonsils. *Collateral Finding:* Loss of motion L. vocal cord. Medical department makes diagnosis of probable T. B.

J. H., male, age 21 years. Cannot clear his throat of secretion. *Diagnosis:* Acute rhinitis and pharyngitis. *Collateral Finding:* Loss of motion L. vocal cord.

E. S., male, age 35 years. Nasal obstruction all his life. Buzzing left ear four to five years. *Diagnosis:* Nasal polypi. O. M. C. C. *Collateral Finding:* Loss of motion L. cord.

S. W., female, age 46 years. Frequent sore throats for two years, especially on right side. *Diagnosis:* Hypertrophied tonsils. *Collateral Finding:* Loss of motion L. vocal cord.

L. P. male, age 30 years. Sore throat for seven weeks. Pain on swallowing and in ears. Had T. and A. operation. *Diagnosis:* Acute rhinitis and pharyngitis. Adenoid remains. *Collateral Finding:* Loss of motion L. vocal cord.

H. K., male, age 36 years. Tonsillectomy one year ago, but continues to have sore throat, especially on right side. *Diagnosis:* Large infected tonsil stump. *Collateral Finding:* Loss of motion L. vocal cord.

Group 2: Patients, Not Hoarse, with Loss of Motion of One Vocal Cord, Having Visible Pathology Elsewhere Suggesting Cause.

J. F., male, age 34 years. Left ear clogged for two months. Had operation for glands of neck many years ago. Tonsils removed. *Diagnosis:* Deviated septum, acute rhinitis, right and left maxillary sinusitis. *Collateral Finding:* Loss of motion L. vocal cord; cicatrices on neck.

O. C., female, age 38 years. Nose fractured 12 days ago. *Diagnosis:* Fracture of nasal bones and deviation of septum. *Collateral Finding:* Loss of motion L. vocal cord. Cicatrices on neck from thyroid operation.

I. A. male, age 45 years. Headache for eight weeks. *Diagnosis:* Chronic pharyngitis and rhinitis. *Collateral Finding:* Loss of motion L. vocal cord; perforation of septum, Wassermann 4+.

V. C., female, age 25 years. Swelling on front of neck. *Diagnosis:* Thyroid enlargement. *Collateral Finding:* Loss of motion L. vocal cord.

Group 3: Patients Who Were Hoarse, with Loss of Motion of One Vocal Cord.

H. A., male, age 38 years. Hoarse for three days. Nasal obstruction, and noise in both ears. *Diagnosis:* Acute rhinitis, pharyngitis and laryngitis. O. M. C. C. *Laryngeal Finding:* Loss of motion L. vocal cord.

H. D., male, age 40 years. Hoarse for one year. *Diagnosis:* Chronic laryngitis. *Laryngeal Finding:* Loss of motion L. vocal cord. Interary thickenings. Medical department reports fibrosis in lungs.

T., male, age 43 years. Hoarse for two months. *Diagnosis:* Subchronic laryngitis. *Laryngeal Finding:* Loss of motion R. vocal cord. R. arytenoid infiltrated. Medical department reports T. B.

It will be seen that the largest group of the 20 cases here reported, 13 in number, comprised all those patients who came for the relief of conditions unrelated to the larynx; *i. e.*, they were not hoarse.

The probable origin of the vocal cord paralysis was, with one exception, not determined at this time. One case was returned by the medical department with the diagnosis of T. B. The second group, four in number, also came for the relief of conditions unrelated to the larynx, but in these we had so to speak a visible clue to the cause, to-wit, trauma following operations on the neck in two; evidence of lues; thyroid enlargement.

The third and last group of three was frankly hoarse and came for the relief of this condition. Two of these had other suggestive appearances in the larynx besides the palsy of the cord, and examination of the lungs in both disclosed lesions, healed in one and more active in the other. The third of this group had an intercurrent acute laryngitis to account for the present dysphonia.

The age of all the 20 ranged from 14 to 47 years, most of them being young adults or persons in early middle age. There were but three women. As you have heard from these histories, the whole range of the commoner diseases of ear, throat and nose were found in these patients. None of the local pathology can be said to have any direct bearing on the laryngeal lesion under discussion. My conclusions may then be summed up under the following heads:

1. Paralysis of the recurrent laryngeal nerve is frequently observed. The assemblage of so many cases during a comparatively short period gives sufficient basis for this claim. One or two patients with this lesion were discovered at almost every session of the clinic.

2. The anatomy of the nerves is to a great extent responsible for their involvement. Under this head I cannot adduce as much confirming evidence at this time as I would like, because a general examination had not been made of all the patients. The fact of frequency is, however, in a way *prima facie* evidence and points to a peculiar vulnerability of the nerves. This thesis of faulty anatomical arrangement of the nerves is given additional force when it is remembered that the longer and more exposed left nerve was involved 19 times while the shorter right suffered only once.

3. Paralysis of the recurrent, partial or complete, and particularly in the early stages, is frequently overlooked. On this score it must be said that none of the cases without hoarseness had progressed to the stage where the laryngeal changes were striking; that is, easily recognized. I doubt whether any or most of them could have been diagnosed except by the method of examination before described.

4. In the absence of hoarseness the patient is unaware of his laryngeal condition. This apparently obvious statement is emphasized because question will arise whether or not it is to the patient's best

interest to disclose the unexpected finding. To my mind this can be best answered after the complete survey of the patient has been undertaken.

5. Paralysis of the vocal cord may serve as the earliest index of a disease elsewhere than in the larynx, such disease having at the time given no discomfort or given rise to any pronounced symptoms. The complete examination of a patient found to have this nerve lesion may disclose any of the intrathoracic pathologies commonly described as causes. A constitutional disorder may be found. The cause may remain entirely obscure, and there are many that fall into this bracket. Some patients may, however, go along serenely for years if the causal pathology is not of vital significance. Such a one may well be left without knowledge of his disability; in any event, we have only then done our full duty by all these patients, whether hoarse or not, when we have summoned to our aid all the resources of diagnosis.

171 West 71st Street.

TERTIARY SYPHILIS OF THE LARYNX.*

DR. LEOPOLD GLUSHAK, New York.

Voice disturbances of varying degrees form the most common coercion as a subjective factor to search the advice of the laryngologist. The extent of voice alteration as a pathological manifestation, though to date scientifically immeasurable, has obtained only popular description and estimation, through interpretation of the impressions on the sensitive ear of the observer. Thus we speak of the raucous voice of the syphilitic, the low and weak voice of the tuberculous, and the husky voice produced by chronic simple laryngitis or associated with abuse of voice. These terms are not scientific determinants and these interpretations by no means pathognomonic of specific pathologic conditions. Yet, a sensitive appreciation of variations in voice dysfunction on the part of the laryngologist may be considered a talent second to his ability in laryngoscopic observation and interpretations of intralaryngeal appearances.

Laryngeal pathology may be an expression of constitutional as well as purely local disturbance. The naked eye appearances as reflected in the laryngeal mirror not only vary according to the type, such as ulcerations, infiltrations, tumors and arthritic involvements, but very frequently remote entities resemble each other and even coexist, such as tuberculosis and syphilis, and carcinoma, syphilis and tuberculosis. It is readily understood that in the discrimination and diagnosis of the separate diseases one must call upon all diagnostic aids: and whereas the history, X-ray findings and blood tests form often the determining factors, yet the clinical interpretation based on visual observation still retains the most important position in the gamut of diagnostic ability. Thus there are most often definite characteristic appearances which direct the mind of the laryngologist to follow certain tracks of reasoning toward correct diagnosis and with all other tests negative will direct him to institute therapeutic procedures in a logical direction. Thus a negative Wassermann may still not move a laryngologist to deviate from diagnosing lues of the larynx when the appearances point that way.

The following case of luetic laryngitis is reported with the intention of registering certain clinical appearances which display

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In respiration.

In phonation. Note the passing of the right cord under the left mass.

characteristic delineations leading to a suggestion of the correct diagnosis.

J. B., age 47 years, came to the Post-Graduate Hospital Laryngological Clinic complaining of hoarseness of six months' duration, for which he was treated for so-called "sinuses." He was told that he had a chronic catarrh of his larynx which would improve on clearing up of his sinus affection. His general physical status revealed a healthy individual with no other complaints but hoarseness. The tone was almost aphonic, gruff and raucous on endeavoring to phonate. His family history was negative. Circulatory system was normal. Chest examination—X-ray—showed a moderate old fibrous thickening at hilus and root, otherwise normal. Urine was normal. Nasal examination revealed a resected septum with a traumatic perforation. His tonsils had been removed; a few residues reappeared. Laryngoscopic examination showed an extensive edema around the left arytenoid, merging with a spindle-shaped swelling in front of it involving the entire false cord and so overlapping the true cord that it could not be seen. Perhaps the swelling extended down to the true cord. This swelling was brownish-red in color, surface smooth and regular. The edema of the arytenoid fixed the left side so that no movement on that side of the larynx was possible. There was a slight edema extending over the interarytenoid membrane and even on to the right arytenoid, on which this edema had no influence as far as motion was concerned. The right side of the larynx showed a dusky color. The vocal cord was somewhat mottled but perfectly free and otherwise normal. On movement the right cord passed under the mass described over the left cord. The edema around the left arytenoid was pinkish in color, in contradistinction to the acute edema which is usually greyish and transparent. There was no pain on swallowing.

The diagnosis on this clinical manifestation was at first lues with a possibility of it being carcinoma of the false cord with inflammatory complication extending to the arytenoid. Wassermann was ordered and returned 4+. Patient was referred to the syphilitic department, where a more detailed history was brought out, including venereal affection 18 years ago without any medication following; he denied secondaries. He was immediately put on treatment and received bismuth and neosalvarsan, to which his laryngeal condition responded promptly, so that in less than one month the hoarseness was greatly reduced, his vocal cords approximated well and the reddening greatly subsided.

This case is reported to present laryngoscopic manifestations on which a diagnosis was made, which, even in face of a negative Wassermann, would have tempted the laryngologist to administer antiluetic treatment as a therapeutic trial. The points which led one to think of lues were the gradual onset and increasing hoarseness, the chronic edema around the arytenoid, the fixation of the cricoarytenoid joint, the pinkish appearance of the edema pointing to chronicity, the smooth swelling involving the left false cord, the painlessness to deglutition. These differentiate the condition from tuberculosis, carcinoma, and rheumatism; and lead one to consider the case as a luetic arthritis of the cricoarytenoid joint with possible gummatous infiltration of the false cord.

1 West 85th Street.

STUDIES IN SINUS INFECTION.*

Dr. J. Wynne Pugh, New York

My studies may be regarded as subject to the well known scholastic Caesarian operation of division into three parts: *1.* A bacteriological and pathological investigation of the already infected sinus, as found freely in any ear, nose and throat clinic in this city. *2.* To search for the predisposing causes for such infections and if possible to try out experimentally any plausible theories of such causation. *3.* And this was mostly to satisfy my own private curiosity, a dredging of medical and allied literature for an explanation of the purpose for which the paranasal sinuses were originally intended.

To deal with the bacteriological investigation first: I sought in this investigation to correlate the bacteria recovered with the reaction of the diseased sinus. Just as in gynecology, the acid-alkaline reaction of the vagina controls its fauna and flora, so it might be possible to find a similar relationship in the mucus fields of the upper respiratory tract.

The specimens were obtained by cleansing the nares and the nasal vestibule, anesthetizing in the usual manner, and then either sucking out the sinus content with the Watson-Williams' technique, or irrigating through a sterile Coakley cannula and catching the outflow in a sterile receiver, the purulent matter being immediately taken up in a sterile syringe and placed in culture tubes.

The Watson-Williams' technique of aspirating into a sterile syringe is excellent, so long as the sinus content is fluid, but I found so many of the sinuses had firm masses of mucopus that the second technique was of more practical use.

The reaction was measured in terms of the hydrogen ion concentration, expressed in terms of pH as devised by Sorensen. The pH was measured by the colorimetric method, three indicators of overlapping range being used, readings with two indicators being taken in each estimation; the indicators used were: *1.* Brom cresol purple, range of pH, 5.3-7.2. *2.* Brom thymol blue, range of pH, 6.15-8.05. *3.* Phenol red, range of pH, 6.75-8.65.

The solution used in irrigating the sinuses under investigation was a sterile isotonic saline. This was chosen after much trial as it was found to be the least liable to distort the pH of the sinus content under investigation. Incidentally, an interesting phenomenon

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may be pointed out here: that there is an amazing difference in the discomfort entailed by the use of various solutions, a strictly isotonic saline at body temperature being easily the most tolerable; any variation in temperature or concentration causing a marked and disproportionate increase in the discomfort, both in the irrigation and the after-behavior of the sinus. In this phenomenon may be found the explanation of the failure in practice of several antiseptic lotions that are successfully used in other fields of surgery, in that they irritate the extremely sensitive sinus mucosa and so defeat their own aim of ameliorating the inflammation.

In all, 101 cases of sinus disease were investigated, and 135 cultures were taken therefrom. The statistics deduced from a study of these cases show the following figures:

BACTERIAL FINDINGS, WITH pH ESTIMATIONS.

Number of cases examined	101
Number of cultures taken	135
Number of pure cultures	37
Number of mixed cultures	98

(A). BACTERIA FOUND IN PURE CULTURE.

	No. of Cases	*Average pH.*	*Range of pH.*
1. Pneumococcus— Type III—4 Type IV—3 Type I—1	8	7.5	7.3-7.8
2. Hemolytic streptococcus	8	7.2	6.5-7.6
3. Hemolytic staph. aureus	7	7.5	7.3-7.6
4. Streptococcus viridans	5	7.5	7.1-7.8
5. Nonhemo. staph. aureus	4	7.4	6.9-7.7
6. Non-hemo. staph. albus	2	7.2	6.8-7.6
7. Non-hemo. streptococcus	1	6.9	-
8. Pfeiffer's bacillus	1	7.5	-
9. Diphtheroid bacilli	1	7.4	-
	—		-
	37		-

(B). BACTERIA FOUND IN ASSOCIATION WITH ONE OR MORE OTHERS.

	No. of Cases	*Average pH.*	*Range of pH.*
1. Streptococcus viridans	39	7.6	6.7-8.1
2. Non-hemolytic staph. albus	24	7.5	6.7-8-1
3. Non-hemo. staph. aureus	19	7.6	6.8-8.1
4. Hemolytic streptococcus	15	7.3	6.5-7.9
5. Non-hemo. streptococcus	15	7.4	6.7-8.4
6. Pfeiffer's bacillus	14	7.5	6.9-8.1
7. Hemolytic staph. aureus	12	7.5	6.7-8.1
8. Hemolytic staph. albus	11	7.6	7.2-8.4
9. Pneumococcus— Type III—7 Type I—1. Type II—1 Type IV—1	10	7.4	6.9-8.1
10. Friedlander's bacillus	7	7.5	6.8-7.9
11. Micrococcus catarrhalis	7	7.5	6.7-8.0
12. Gram negative diplococci	6	7.5	7.2-7.6
13. Micrococcus Flavius	4	7.4	6.8-7.9
14. Gram negative bacilli	3	7.3	6.9-7.7
15. Diphtheroid bacilli	2	7.25	7.2-7.3
16. Micrococcus fecalis	1	8.1	-

These were obtained from 98 cultures and may, therefore, be regarded as approximately the percentage figures as well. From these figures one may deduce the thoroughly satisfactory conclusion that the range of hydrogen ion concentration compatible with a full, happy and useful life for all the bacteria recovered is identical with that of human cell life, and that no bacteria thrive especially, or are unusually discouraged, in any one pH.

But other conclusions are drawn from further study of the same cases which are somewhat unusual, and one of them rather startling in its implication.

The first is the correlation between the type of sinus content and its reaction. Almost always the foul, broken-down, cloudy return from an acute or recrudescent sinusitis has an acid reaction relative to the normal healthy average pH of 7.5, the pH varying from 6.9-7.4. On the other hand, the healing sinus, which tends to have a firm clot of mucopus, has a pH varying from 7.5-8.1; so that the use of the alkaline lotion in the irrigation of infected sinuses may have biochemical as well as mechanical reasons for its employment.

The other and more startling discovery was that the bacterial flora of a diseased sinus changes continually. Several of the cases investigated had cultures and pH estimations at intervals of one-two weeks, and it was decidedly the exception to find the same bacteria present at different examinations.

For example, in the case of R. B. Y.: Oct. 7, 1930: Plugs of mucopus, pH 7.4; nonhemolytic streptococci; staphylococcus aureus. Oct. 21: Large masses of mucopus, pH 7.6; hemolytic streptococci; nonhemolytic streptococci; Pfeiffer's bacillus. Dec. 9: Broken-down purulent matter, pH. 6.9; hemolytic staphylococcus aureus; Pfeiffer's bacillus.

And again in the case of P. C.: Nov. 3, 1930: Foul-smelling green pus, pH. 7.6; streptococcus viridans. Nov. 10: Broken-down, cloudy return, pH 7.2; pneumococcus Type III. Nov. 19: Foul, brown debris, pH 6.7; streptococcus viridans; hemolytic staphylococcus aureus.

Several other cases could be quoted to demonstrate this point, and its importance is seen when one considers the preparation of autogenous vaccines. Pathologically, the excellent sections shown in the Archives of Otolaryngology for February of this year, in the article by Dr. Kistner, show the progressive infiltration of the sinus mucosa by bacteria far better than any slides that I have been able to prepare. Seeing such sections, and considering the bacteriology of the healthy mucous membrane, one may well visualize how tran-

sient invaders may be recovered even in pure culture from the sinus cavity while the real cause of the trouble, embedded in mucosa, evades detection. I would suggest that in the preparation of autogenous vaccines repeated cultures be taken first, and that then only those vaccines be used which prove their specificity by the degree of reaction they cause on injection in trial doses.

I think that will do for the main part of my discourse, and as my time is nearly over I shall refer but lightly to the other two parts of this but partially conquered Gaul.

2. This is the problem of the predisposing cause of sinus trouble. I am British, and I think that we have very much less sinus trouble than you do. We also have a much more moist climate, aquatically speaking, and a healthy dislike of central heating. There is a well established tradition, even over here, that central heating is the cause of a good deal of sinusitis; but so far there is no proof. It seems to me it might be possible to humidify some public schools and to use nonhumidified scholars as controls. Alternately, rabbits could be subjected to similar investigations; that is, if the antivivisectionists would permit their use instead of school children, for rabbits have magnificent frontal and maxillary sinuses, and in captivity are very prone to such infection, the condition being known as "snuffles." This leads to a septic bronchopneumonia and quite often death.

Once given an abnormal mucosa, whether by atmospheric or infective influences, one can easily visualize the paranasal sinuses temporarily shut off from ventilation and becoming incubators for any bacteria that happen to be enclosed therein.

3. As for the reason why we have sinuses at all, I can do no more than quote the several theories that have been encountered in the literature, and leave their evaluation to the discrimination of the individual:

A. That in the more primitive animals the paranasal sinuses were developed as a means of extending the area of the olfactory mucosa, and that the sinuses in man are vestiges which have acquired other physiological functions.

B. That the sinuses assist the nasal mucosa in moistening and warming the inspired air. This view is supported by the provable variations in pressure within the sinuses, varying with the phases of respiration, and showing the existence of a current of air.

C. That the rarefaction of the bones in the front part of the skull is of assistance in giving the skull the optimum degree of equipoise.

D. That the light, elastic character of the bones surrounding the orbit and the anteroinferior aspect of the cranium is of definite function as protecting these delicate structures against shock, the sinuses being the means whereby these bony laminae are formed.

E. In the lowest amphibians one observes the development of sinuses in the air-breathing forms. Possibly this is a mechanism to insure that the respiratory orifice shall be uppermost when the animal is submerged in water. The situation of the sinuses in all animals, even in men and monkeys, would facilitate this function. It is interesting to note that in those mammals that have reverted to a predominantly aquatic life, and whose respiratory apparatus is therefore less liable to unexpected flooding, the sinuses tend to disappear altogether in the adults; this has actually happened in the seal and walrus families.

F. Older physicians advanced the view that the sinuses form resonating chambers for the voice, but it is now doubted whether this is more than a very secondary function, if of any value at all.

I have refrained deliberately from quoting any references or authorities as that would serve merely to pile Tedium on Boredom; but I must express my sincere gratitude to Dr. Coakley, Dr. Babcock and my colleagues at the Medical Centre. Without their help, advice and criticism you would not now be gaining your incipient rest from this paper.

Presbyterian Medical Centre.

FOREIGN BODIES IN AIR PASSAGES.*

DR. GEORGE R. BRIGHTON, New York.

Since the perfection of the distally lighted bronchoscope by Dr. Chevalier Jackson, the successful removal of foreign bodies from the air and food passages has become more or less common among endoscopists. The following two cases are presented here, first, because they are of interest from the standpoint of their history; and, second, because both had the same type of foreign body as the causative factor of their symptoms; namely, a safety pin.

Case 1: Mrs. S., age 31 years. Admitted to Eye, Nose and Throat Service of Presbyterian Hospital on Oct. 27, 1929. *Chief Complaint:* Swallowed a safety pin.

Present Illness: Patient reported that four days previously she was diapering her baby and put a pin in her mouth and inadvertently swallowed it. She tried to reach it with her finger but was unable to do so. She has had a moderate amount of discomfort in the back between the scapula and in the region of the manubrium. This discomfort was exaggerated on coughing and swallowing. Soon after swallowing pin, patient expectorated some blood. The remainder of the history was not relevant.

On admission, patient's temperature was 101°, pulse 100, respiration 24. Fluoroscopy showed a safety pin in the right side of the lung. Stereoscopic X-ray plates were made at once, showing the safety pin just below the bifurcation in the right bronchus, point up, the keeper lying near the opening of the upper lobe bronchus.

Under local anesthesia, 10 per cent cocain to the superior laryngeal nerves, a nine m.m. bronchoscope was easily passed down to the carina. The point of the pin was visualized and brought into the scope by means of a Tucker tack and pin forceps. It was noted that the keeper was in the upper lobe bronchus, so that the whole pin had to be gently pushed downward into the right bronchus, bringing the keeper into view. It was then a simple matter, with the point in the bronchoscope, to bring the keeper into the large lumen of the instrument.

The patient had a moderate temperature reaction the next day, the 4 p. m. temperature being 102.8°, but this quickly subsided and she was discharged on the fifth day. Since then she had had no symptoms referable to her chest. Check-up X-ray after removal of foreign body showed no lung changes.

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Case 2: Baby A., female, age 7 months. Admitted to Babies' Hospital, Aug. 5, 1930. *Chief Complaint:* Coughing and hoarseness since May 1; three months.

Present Illness: About three months previous to admission child apparently had a cold, considerable cough and a very hoarse cry. No temperature. Was taken to a physician, who made a diagnosis of pneumonia.

X-ray of chest was taken on May 13. Diagnosis of enlarged thymus made. For next six weeks patient received X-ray treatments for the thymus. The cough gradually improved during treatment, but hoarse cry continued. Two weeks after the final X-ray treatment the symptoms returned as before. The child was taken to another physician, Aug. 4, and X-rays of the chest were made in two planes and a safety pin was seen in the sagittal plane in the region of the larynx. Admitted, Aug. 8, for removal of foreign body.

Under direct laryngoscopy a safety pin was seen in the subglottic space. There was very little reaction above the cords, except a slight redness. The keeper was in the anterior commissure; point was not visualized. In order to bring the point into view the keeper was grasped and gentle pressure was made in a downward direction. The pin broke off at the spring and this portion was easily removed. The pin was found very much corroded, in several areas almost completely oxidized.

Inasmuch as the patient was in an acutely inclined position, with the head down, there was little danger of the remaining portion of the pin going down into the lung. Remaining portion was visualized and lower end grasped in forceps and with gentle pressure downward the point was released from the granulations and the pin removed.

There was no bleeding from any of these manipulations; the child's condition and respiration were good so it was deemed advisable not to do a tracheotomy.

The case was carefully watched by a well trained nurse and one hour after the operation the child was sleeping comfortably, with no disturbance in respiration.

Patient was discharged on the second day postoperative and has been well ever since, except for an attack of measles.

CONCLUSIONS.

1. In any case of unexplained cough or pain in the chest, careful X-ray in both anteroposterior and lateral planes should be made.

2. In all children any symptoms pointing to the air passages should be considered due to foreign bodies until proved otherwise.

580 Park Avenue.

PRIMARY JUGULAR BULB THROMBOSIS—METASTATIC ABSCESSES; REPORT OF CASE.*

DR. HERMAN F. LAMPE, New York.

The general consensus of opinion as to the cause of primary jugular bulb thrombosis is that the infection enters the bulb through a dehiscence in the floor of the tympanic cavity. A thrombus may form through this pathway without causing any particular changes within the mastoid.

A boy, M. F., age 13 years, was admitted to the service of Dr. T. L. Saunders at the New York Eye and Ear Infirmary on Aug. 3, 1928, with a temperature of 105°.

Past History: Irrelevant. *Present History:* Nine days prior to admission he had complained of a pain in the left ear following a head cold. Five days later a discharge resulted. No history of a chill was obtained.

Findings: A general physical examination showed that of a slender male, face flushed and appearing to be very ill. Heart, lungs and nervous system were negative. Throat showed moderately hypertrophied tonsils with no inflammation present. Nose was negative. The right ear was normal and the left showed a perforation in the posterior inferior quadrant of the tympanic membrane, through which a very thin watery discharge exuded. There was no sagging or bulging present. There was some enlargement of the cervical glands, but no mastoid tenderness present. The X-ray at this time showed a pneumatic mastoid, hazy throughout, but the detail good. A blood count and culture were made and the count showed 4,500,000 red cells, 12,200 whites, 90 per cent hemoglobin, 17 per cent small mononuclears, 1 per cent large mononuclears, and 82 per cent polymorphonuclears.

Operative Procedure: The same day a simple mastoidectomy was done and the bone was found congested throughout, but no pus or granulations were present. The lateral sinus was exposed intentionally over a rather large area at the knee but appeared normal.

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The following day the temperature went to 106° and the report from the blood culture showed numerous colonies of hemolytic streptococci in 20 hours.

The child was again taken to the operating room, the mastoid reopened and a larger area of sinus exposed. Towards the torcular end it was perfectly normal, but close to the bulb it assumed a grayish appearance and pus was encountered just at the bulb. The vein was then resected for its entire length and was found to be completely clotted. The sinus was then opened by the usual method and free bleeding obtained immediately from the torcular end. It was difficult to get bleeding from the bulb, so a small suction tube was inserted and after a large amount of pus was removed, profuse bleeding followed. Both ends were packed with iodoform and the usual dressings applied. The following day a blood transfusion was given by Dr. Stetson, 500 c.c. being given in all. The patient was semicomatose before the transfusion but seemed to rally afterwards. Four blood-transfusions were given in all.

Complications: The temperature continued to range between 103° and 106°. At this time the patient complained of pain in the right wrist. This became red and swollen, and was opened through the articular capsule by Dr. McGowan. A large amount of pus was drained from here. Two days later there was pain over the right side of the chest and a diagnosis of empyema was made by Dr. Irving. Aspiration produced 35 c.c. of a seropurulent fluid. The following day the temperature dropped to 102° and varied between this and 100° until the fourteenth day after admission, at which time it reached normal and remained so thereafter.

Result: The leukocyte count went as high as 20,250 at the time of the empyema. The patient remained in the hospital for one month, then was transferred to the clinic for further dressings. The mastoid and neck wound healed rapidly. Likewise the empyema and wrist infection gave no further trouble.

34 East 51st Street.

THE REPORT OF OPERATION FOR THE REMOVAL OF AN EXOSTOSIS FROM THE POSTERO-INFERIOR WALL OF THE EXTERNAL AUDITORY CANAL.

Dr. J. Warren White, Norfolk, Va.

Exostoses spring from the bony portion of the canal external to the drum and are frequently attached to the drum. Many etiological factors have been assigned but it is assumed that the exostose originate from a variety of causes. These growths are benign and are usually multiple. They rarely occlude the canal entirely or impair the hearing.

My report refers entirely to the growth with a sessile base and not to the pedunculated variety which can be easily pried out. The textbooks do not speak of any serious complications in the operation for removal of exostoses of this character, but advise a curved post-auricular incision and separating the membranocartilaginous meatus from the bony canal. Then with a small chisel or gouge remove the bony growth.

This being my first attempt at this particular operation, I expected to have no difficulty. Since performing this operation, my second attempt will be made with considerably more hesitancy. In looking up the literature on this subject to see if someone else had not experienced some difficulties, I came across an article in The Laryngoscope, August, 1929, by Dr. J. R. Page. After reading this article I felt that I had been extremely fortunate. Dr. Page says: "One has but to attempt this operation to discover the difficulties of its after-treatment and the almost inevitable disappointment that results and his conclusion is that when exostoses of the external auditory canal have so occluded the canal that the operation for their removal is indicated, the modified radical operation with complete exenteration of the mastoid offers the most satisfactory solution of the problem."

A woman, age 30 years, was referred to me with what was thought to be a furunculosis of the external auditory canal of the left ear. She gave a history of being told, when a child, that there was an obstruction in the ear and that it would be dangerous if an

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infection developed in the middle ear, left side. She complained of dizziness at times and an increasing deafness in the left ear. Periodical attacks of pain in the left ear, lasting for several days, were becoming more frequent.

On examination I found an exostosis arising from the posteroinferior wall. The growth completely blocked the canal and there was considerable inflammation and pain caused by the epithelial debris and moisture collected in the canal.

After local treatment over a period of several weeks the inflammation subsided. I hesitated at first advising the operation, but even upon suggesting operation she immediately wanted it done. As she was very insistent and finding nothing in the textbooks calling attention to the complications, I advised removing the exostosis. If the patient had been apprised of the complications I would have felt better, but she was admitted to the hospital, expecting to have a small operation and be out in a few days.

Under gas anesthesia a curved postauricular incision was made over the mastoid and the auricle was carried forward, then separating carefully the membranous canal from the posterior wall. It was impossible even with the greatest care to separate the lining of the canal from the exostosis on the posteroinferior wall without tearing it. Any traumatism of the anterior wall may result in an atresia of the canal and deep adhesions between the two walls. The bony growth was removed with a small chisel and a sharp bone curette, which was introduced and removed from within outward. The bone was ivory-hard and care had to be taken not to invade the subcortical cells of the mastoid. The danger we know would result in secondary infection of the mastoid and eventually would require a mastoidectomy. It took me one hour of hard work to accomplish what I thought was going to be a very simple procedure. The drum membrane was not injured and the anterior wall was not traumatized or disturbed. The postauricular incision was closed with interrupted sutures. The posterior canal lining was very much torn and the remnants were replaced in the excavated posterior bony wall and held in place by gauze packing in the canal. She had no pain or complications following the operation. The stitches were removed on the third day.

On the fourth day she suffered severe pain in the ear, and on examination I found so much swelling in the canal that I could get no idea what was going on in the middle ear. The next day the ear began to discharge but the pain persisted. I saw her twice daily for a while and each day I expected to readmit her to the hospital for

a mastoidectomy. After one week of anxiety on my part she finally recovered without any further operative interference. The final result was all that I could expect. The canal was normal in size and there was no difficulty in introducing a large ear speculum.

My conclusion is that exostoses with a sessile base should not be removed unless giving definite symptoms that make their removal imperative, and as it is considered a minor procedure the patient should be warned of the complications that may develop.

816 Medical Arts Building.

TWO UNUSUAL CASES OF LABYRINTHITIS.*

DR. CHARLES MANLEY GRIFFITHS, New York.

These two cases happened to be in the Manhattan Eye, Ear, Nose and Throat Hospital at the same time and illustrate acute labyrinthitis complicating, first, an acute mastoiditis and, second, an operation for chronic mastoiditis.

Case 1: A white male, age 26 years, admitted to the hospital, Oct. 31, 1930, complaining of slight pain, discharge and swelling behind right ear, with some dizziness. The family and past history seemed unimportant. No previous ear symptoms and says his hearing was good before onset of present illness.

Present Illness: Three months previously he developed a severe pain in right ear following a cold. A day or so later there was a spontaneous discharge, which has persisted to the present time. About three weeks ago he had a series of dizzy attacks with nausea, but no vomiting. Did not fall. This has gradually lessened and is now only brought on by sudden movements. About a week ago slight swelling developed behind the ear. Examination: Well nourished man. Temperature, 100.2°. Heart, lungs and abdomen, normal. Left ear, normal. There is a moderate subperiosteal swelling over right mastoid, which is slightly tender. Small amount of thick, purulent material in right canal, which is markedly collapsed.

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Functional: No spontaneous nystagmus. Patient tends to fall to the right. Hearing tests: Left normal. Caloric test normal, right; no hearing could be demonstrated with noise apparatus in left ear. Caloric test: No reaction after five minutes' irrigation with water at 68°. The X-ray report was as follows: Left mastoid, pneumatic and clear. Right mastoid, pneumatic; severe involvement with marked absorption. A diagnosis of acute diffuse labyrinthitis in the quiescent stage was made and it was decided to do only a simple mastoidectomy.

Nov. 3, usual operation was performed, avoiding, however, the use of the gouge and mallet. The cells were found to be broken down and the sinus exposed and covered with dense granulations throughout. No fistula was found. The patient was kept in bed for one week, but there was no postoperative reaction, the temperature never going above 99.8°. Healing was rapid and the patient was discharged from the hospital, Nov. 17, with an intact membrane and a practically healed mastoid wound. Patient was seen a few months later, when there was still a complete loss of cochlear and vestibular function, but a completely healed ear; and he was suffering no inconvenience from the inactive organ.

Case 2: A white girl, age 19 years, admitted to the hospital, Oct. 22, 1930, complaining of discharge from the right ear for one-and-a-half years. *Past History:* Good general health, no ear symptoms until onset of present illness.

Present Illness: Eighteen months ago developed pain and discharge after swimming. Has had almost constant treatment since that time but the discharge has persisted. There is no pain and deafness is not complained of.

Examination: Exceptionally well nourished young woman, with normal heart, lungs and abdomen; temperature 99.6°. Left ear normal on inspection. Right ear, moderate amount of discharge, with some odor. There is a small marginal perforation in upper posterior quadrant, otherwise the drum is intact. No mastoid tenderness. Functional examination: Hearing in left is very acute; right is diminished about one-half but retains decidedly useful hearing. Whispered voice heard at about 10 feet. There was no spontaneous nystagmus. Fistula test negative and the caloric reaction was normal in both ears. X-ray report: Sclerotic mastoid. Some absorption near knee of sinus. On account of the relatively good hearing it was decided to do a modified radical mastoidectomy. Oct. 24, the usual operation was done. The mastoid was partially sclerotic but considerable softening was found. The postcanal wall was lowered and

flaps cut and sutured in the regular way. There was a postoperative rise in temperature to 100.8°, which then declined to 98.6, and the course was uneventful until the night of Oct. 30, six days after operation, when the patient complained of some headache, dizziness, nausea and vomiting. The temperature was only slightly elevated, 100.4°. Examination the next day, Oct. 31, patient lies in bed on left side, left ear partially covered, extreme nausea, frequent vomiting, marked combined nystagmus to the left. With noise apparatus in left ear no hearing could be demonstrated. A diagnosis of acute diffuse labyrinthitis with destruction of right cochlear and vestibular function was made, and the patient was kept as quiet as possible. Sedatives and fluids by rectum given. All symptoms gradually subsided. Nov. 5, very slight nystagmus, hearing absent on right, on walking falls to right, no headache. Improvement continued until discharged from hospital, Nov. 19. Dressings were continued in clinic, the membrana tympani healed completely and the cavity epithelialized perfectly. She has been observed at intervals until the present, the cavity and drum membrane have remained dry, but there has been no return of cochlear or vestibular function.

These two cases are of interest primarily on account of the etiology. In Case 1 the labyrinthine destruction apparently occurred during the course of an acute suppuration, with unusually mild symptoms, and resulted in a perfect walling off of the infection by Nature, so that a simple mastoidectomy produced a cure of the suppuration.

In Case 2 a labyrinthitis develops six days after operation, which seems too long to be the result of a surgical accident, and in any event dislocation of the stapes seems improbable in a modified radical. On the other hand, if it is simply an extension or an aggravation of the pre-existing infection, it would seem unusual to have a prompt and complete healing of the drum membrane and mastoid cavity.

114 E. 54th Street.

A RARE PATHOLOGIC-ANATOMIC FINDING.*

Dr. V. Gakkel and Dr. A. Minkovsky, Leningrad U. S. S. R.

When autopsy reveals syphilitic lesions of the organism, scars of a luetic origin are not infrequently also found in the trachea. However, a syphilitic gumma of the trachea is a far less common occurrence. Thus, the list of autopsies (over 11,000 cases) performed by the histopathologic section of Mechinkov's Hospital registers but one such case. Harmer[1], as well as a number of other authors, has stated that lues occur in the trachea less frequently than in the larynx.

Consequently an isolated syphilitic lesion of the trachea without any simultaneous manifestations of the disease in the larynx may be regarded as a true pathologicoanatomic rarity.

The case we further describe may prove to be of particular interest from this point of view.

The patient, A., male, unskilled worker, age 30 years, was admitted to the hospital on Dec. 27, 1929, on account of dyspnea. The patient complained of dry cough, occasionally attended with expectoration, and of pains in the left side of the thorax. According to anamnesis the patient had never suffered from tuberculosis. The history of the disease showed that in June, 1929, the patient had a swelling of the neck, which opened in July, discharging a large quantity of pus. At the time he had been treated with quartz and iodoform injections. A month previous to his entering the hospital he developed dyspnea and a severe cough. Within the last three weeks a swelling of the right ulna had also been observed, which corresponded in size to that of a pigeon's egg. The medical examination performed on his admittance to the hospital revealed that the patient was under-nourished and produced the impression of one dangerously ill. It also revealed a soft swelling of the right ulna and an inconsiderable indentation at the lower edge of the sternum. Accentuation of the second aortal tone. Size of the heart, normal. Breathing was difficult, as in stenosis. The cervical, inguinal and axillar lymphatic glands were enlarged, while palpation revealed their fibrous consistency. There was a fistula found on the neck above the manubrium sterni. Temperature, 36.6° in the morning and 37.8° in the

*From Histopathol. Section of Mechnikov's Hospital in Leningrad.

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evening; pulse rate, 96 per minute. On Jan. 3, 1930, examination revealed multiple compact lymphatic glands on the right humerus, spreading along the whole length of the vascular bundle. X-ray examination on Jan. 3 showed marked shadows of both apices of the lungs; transparent subclavicular region; large groups of lymph-

Fig. 1. Syphilitic gumma of the trachea.

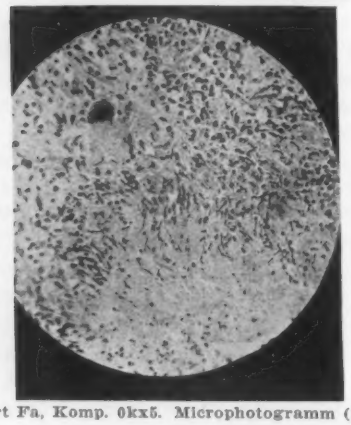

Fig. 2. Reichert Fa, Komp. 0kx5. Microphotogramm (tertiary lues of the trachea).

atic glands in the hilus; dilatation of the peritracheal and mediastinal glands. The lower section of the right lung is not clearly outlined. On Jan. 4 the patient complained of troublesome cough, stenotic respiration and restless sleep. Wassermann reaction clearly positive (4+). Jan. 5, severe cough and dyspnea. Jan 6, st. idem; Jan. 8, tracheotomia in extremis under local anesthesia. Exitus.

Pathologicoanatomic Diagnosis: Tertiary syphilis of the trachea, complicated by stenosis. Brnochopneumonia- catarrhalis. Syphilitic scars of the liver and the spleen (with amyloid degeneration). Mesaortitis luetica. Extracts from the pathologicoanatomic minutes: the peritracheal lymphatic nodules present on the surface of the incision are compact and of a gray-reddish color; the peritracheal glands are somewhat enlarged, compact and gray in color; the mucosa of the trachea shows deep, extensive ulcerations of an irregularly circular shape; in some places there are partial destructions of the cartilage (see Fig. 1).

The postoperative (tracheal) stoma is surrounded by an ulceration of 6x2 c.m. in size, which has a flat bottom and even edges. The microscopic examination of sections obtained from the edges of the ulcer, from the peribronchial gland, the upper lobe of the right lung, the scars of the liver and the spleen, from the wall of the aorta taken within the region of its arch, provide the following data:

1. The bottom of the ulcer contains a superficial necrotic layer, underlaid by an infiltration consisting of circular cells which contain numerous lymphocytes; the lower layer is of granular and fibrous tissue, penetrated with blood vessels, the latter being surrounded with numerous lymphocytes. There is a large number of newly-formed blood vessels of the capillary type present, consisting of one layer of endothelial cells (see Fig. 2).

2. The bronchial ulcer shows large necrotic areas surrounded by a lymphatic infiltration, where both epithelioid and gigantic cells are to be found. Numerous lymphocytes are dispersed along the blood vessels, the spaces between them containing plasmatic cells. A similar type of necrosis containing giant cells was observed in the peritracheal lymphatic glands, in the scars of the liver and the spleen. The section taken from the wall of the aorta showed a thickening of the intima; small, limited areas of necrotic tissue in the media; numerous lymphocytes in the vasa vasorum and hyalinosis of collagenic fibres. Staining after Bielshovsky revealed a network of gitterfasern within the necrotic areas. Phenolfuchsin staining, negative. Sections taken from various portions of the lung revealed no signs of tuberculosis. Thus, both macro- and microscopic examinations established a manifest syndrome of tertiary syphilis of the trachea.

The case had been clinically diagnosed as tuberculosis; however, the pathologicoanatomic dissection failed to find any symptom which might have confirmed the diagnosis.

Kaufmann[2] states the fact that tracheal syphilis but rarely coincides with bronchial syphilis without a simultaneous affection of the larynx; namely, conditions characteristic of our cases. Therefore, the reported case may be considered to be rare from this latter point of view as well.

The improvements introduced into the methods of examining the upper respiratory passages have resulted in a large number of correctly diagnosed cases of syphilitic lesions of the trachea; thus, the number of cases diagnosed has increased in accordance with the introduction of new methods of investigation. The following table serves to illustrate the above stated fact: In the year 1867 Gerhardt described 22 cases of clinically diagnosed lues of the trachea; in 1871 Vierling reported 40 such cases; not long ago Nicolai presented a description of 352 cases. Mann[4] mentions 31 cases, out of which seven proved to be fatal. Onodi, Pollak, Sack and Killian have also made reports on cases of luetic lesion of the trachea.

Killian believes the right bronchus to be more frequently affected with lues than the left one. However, a typical gumma of the trachea is but rarely revealed by pathologicoanatomic dissection. From the practical viewpoint it appears to be most important to apply a specific therapeutic treatment in every case where stenosis of the trachea is either manifest or may be supposed.

LITERATURE.

1. HARMER, L.: Denker und Kahler, Bd. III, Tl. III, S. 1104.
2. KAUFMANN, H.: Lehrbuch der speziellen pathol. Anatomie, Bd. I, S. 255, 1922.
3. HENKE und LUBARSCH: Handbuch der speziellen Pathologischen Anta. und Histol., Bd. III, Tl. I, S. 410, 1928.
4. MANN: Handbuch von Katz und Blumenfeld.

Gulyarnaya Str. N. 10 C-21.

A SIMPLIFIED METHOD FOR CORRECTION OF DISHFACE.

Dr. Jacques W. Maliniak, New York.

The condition here described is characterized by the recession of the middle part of the face and lower part of the nose, with à prognatic chin.

Description: The most striking feature of a dishface is the disproportion between the prognatic chin and short upper jaw. The malformation of the jaws results in the malarrangement of the teeth. The deformity of the nose is characteristic; the lower part is held backwards, attached to the under-developed upper jaw, thus giving a humped and flattened appearance; the alae are flattened and the nasal apertures narrowed. Preventive treatment is purely orthodontic and must be carried out in early childhood.

Repair of the Constituted Deformity: The depressed middle part of the face around the lower half of the nose must be raised by filling out the deficiency along the lower border of the aperture and the maxilla. A procedure which aims at the correction of this deformity consists in the affixation of a cartilaginous arch along the nasal aperture, thus advancing the depressed region. A cartilaginous transplant is introduced horizontally through an incision in the columella and properly affixed to the periosteum. In addition to this operative step, which aims to raise the depressed paranasal area, a proper reconstruction of the nose is required. The method which we find most suitable for the repair of this deformity follows:

An incision is made on the middle part of the philtrum and slightly continued on the upper lip, through which an undermining of the lip and alae is done. The horizontal tunnelling should not exceed one-half-inch in diameter and extend to the insertion of the alae on each side.

A free rib cartilage graft, about one-third of an inch wide and about three inches long, is removed from the seventh or eighth rib cartilage, leaving the perichondrium attached to the inner surface. The graft is prepared so as to thoroughly preserve the bent appearance approaching the semicircle (Fig. 1-A). In the middle of the arched graft a small opening is made for the insertion of a short cartilaginous graft extending from the anterior nasal spine towards

the tip of the nose (Fig. 1-B). A third graft is placed in the dorsal depression and extends from the tip of the nose to the nasal bones (Fig. 1-C).

The first two grafts, over the maxilla and the columella, should maintain a certain intimacy if they are to fulfill the satisfactory raising of the nasal tip and of the lateral depressions.

The fixation of the horizontal graft is partially accomplished by its exact fitting into the narrow subcutaneous tunnel, and to avoid displacement the graft is attached to the subcutaneous tissue and periosteum with a few buried sutures.

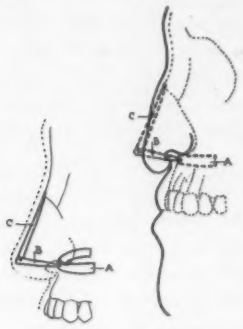

Fig. I.

Fig. 1. Nasal shape in a dishface deformity. "A," "B" and "C"—The three cartilaginous grafts designated to raise the depression of the upper lip, nasal tip and dorsum.

Small diagram: showing the relationship of the cartilages and the corrected deformity.

The proper immobilization of the dorsal cartilage is accomplished by lodging its upper pole in a bony groove (Fig. 1-C). The oversized bony part of the dorsum, often present, is levelled down with a bayonet-shaped saw and the nasal bones separated at the middle line; a groove is thus formed for the insertion and immobilization of the dorsal graft. The excessive length of the nose is reduced by excision of a triangular fragment from the septal cartilage; smaller fragments from the lateral nasal cartilages are excised for the shortening of the lateral walls.

Summary: A new method of correction of the dishface deformity is here described. The dishface deformity is characterized by an under-developed upper jaw with a flattened lower part of the nose. The depressed middle part of the face is raised by the affixation of a cartilaginous arch along the base of the nasal aperture. The graft is introduced through a vertical incision of the columella encroaching upon the upper lip. A second short graft extends from the center of the arch to the nasal tip.

The dorsal depression is corrected by a separate cartilaginous graft extending from the nasal tip to the nasal bones.

Fig. 2A.

Fig. 2B.

Fig. 2A. A typical dishface deformity characterized by recession of the middle part of the face and prognatic chin.

Fig. 2B. Condition after operation; the upper part of the lip and the region of insertion of the alae are raised by a bent cartilaginous graft introduced through a long columellar incision. The nose is shortened and the dorsal depression corrected by a separate graft.

1125 Park Avenue.

NUPERCAIN, THE NEW LOCAL ANESTHETIC, AND ITS USE IN NOSE AND THROAT PRACTICE.*†

DR. A. F. LASZLO, New York.

Constant efforts to eliminate cocain have brought a new product on the market; percain, the name applied in Europe, and used for more than two years, and nupercain, the name given in the United States, and distributed by the Ciba Co. The first favorable report to appear in this country by general surgeons was published by Keyes and McLellan[1]. Since the appearance of their report, many gynocologists and urologists have been using the drug, whose main advantage is the supposedly lasting effect of the anesthesia, six to eight hours after the operation. Up to the present time, no reports have been published in the United States by nose and throat specialists. Beck[2], however, in an article written by him in 1930, mentions the use of nupercain in surgery of the inferior turbinate. Many reports relating to the use of nupercain in our specialty are available by German and Swiss authors.

For the past few months, we have been using nupercain in the operative clinic of the Nose and Throat Department of the New York Post-Graduate Hospital and Medical School, with the possibility of eliminating cocain from nose and throat work.

Nupercain is different from other local anesthetics previously used. Mischer[3], of Basle, in 1929, synthetized this new drug as alpha-butyl-oxycinchonic-acid-diaethyl-ethylen-diamid-hydrochlorid. As we see, nupercain is a chinolin derivative, in the form of colorless crystals, easily soluble in water, may be sterilized without deterioration by boiling, readily mixible with adrenalin and ephedrin, and has no unpleasant taste. The specific gravity of the effective solution may be as low as 1002. It is very sensitive against alkalin, and must be kept in alkalin free glass (jenaer). The saline solution for dissolving must not contain any traces of alkalin (sodium carbonate and bicarbonate); if any traces of alkalin exist, the drug will precipitate. This is a very important factor when the concentration is low. The precipitate may be dissolved again with a few drops of diluted hydro-

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†From the Nose and Throat Department of the New York Post-Graduate Hospital and Medical School.

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chloric acid, about three drops to 1000 c.c. As to the effectiveness of the drug, Ullman[4] shows that 1:125,000 has already an effect on the frog reflexes against cocain 1:10,000, and 1:75,000 has already a paralytic effect against cocain 1:5000. The onset of the anesthesia is a few minutes retarded as compared with novocain, but lasts much longer, especially if the drug is mixed with adrenalin. Even 1:2000 will give an anesthesia, and 1:100 can be used for topical application.

As to its toxicity, opinions differ. Lipschitz and Laubender[5] quote that the toxicity is only twice as much as cocain. Ullman[4] and Ruedi[6] estimate this toxicity as five times as high as, or even higher, than cocain. However, its relative toxicity is smaller as compared with cocain inasmuch as 1:1000 solution gives a good anesthesia for infiltration or conduction, and 1:50 will be equivalent to 5 or 10 per cent cocain for topical application. The lethal dose, according to Ullman and Ruedi, is 0.05 to 0.07 per k.gm. for cocain, 0.40 per k.gm. for novocain and 0.01 per k.gm. for nupercain; and although as we see, the nupercain can be used in 1/10 of 1 per cent solution, it will still be about 20 times as toxic as novocain. And, if we adhere to Braun's principle relating to local anesthetics, we find that the main objection to nupercain is its unfitness to comply with his first rule, namely: The substitute must be less toxic than the drug which is substituted.

The use of nupercain has to be examined from three angles. First, as infiltration or conduction anesthesia. Second, used topically for operative purposes in nose and throat work; and third, as topical application for diagnostic purposes only.

The use of nupercain for infiltration and conduction anesthesia would mean a substitute for novocain. Since Einhorn discovered novocain in 1905, its use has proved that it is an excellent local anesthetic, and although there are reactions and some deaths reported, its discovery is unquestionably the most important advancement in local anesthetics since the discovery of cocain as a local anesthetic by Koller, in 1884. Novocain, in one-half of 1 per cent solution, combined with kal.-sulph. and adrenalin, can be used in almost unlimited quantities, 300 to 500 c.c., without any toxic effect.

Contrary to this, Siebner[7] reports a death with nupercain after using 90 c.c. of 1:1000 solution; Freund[8], 130 c.c. of 1:1000 solution. There were many deaths reported after the use of 1 to 2 per cent solution for injection in spinal anesthesia, where this percentage of the anesthetic was injected by mistake. No idiosyncrasy has been reported against nupercain. As to the local reaction, Albersheim[9] noted more bleeding at tonsillectomies than after the use of novo-

cain, and Ruedi saw sloughing of the palate after an injection of 1:1000 of nupercain. Against this disadvantage, the great advantage would be the more lasting effect of the anesthesia.

My personal experience, which is based upon more than 125 local tonsillectomies up to the present time, has not definitely proven its merit as regards the lasting effect of the anesthesia. When I first started to use nupercain, I injected 1:1000 nupercain on one side, adding adrenalin 1 drop to a dram, injecting on the other side, novocain one-half of 1 per cent, adrenalin 1 drop to a dram, in about 15 cases. I did not notice any difference between the two sides in the bleeding at the operation or postoperative. As to the postoperative pain, there were cases in which the pain was the same on both sides, some cases reporting even less pain on the side which had received the injection of novocain, and some reported no pain for a few hours on the side where the nupercain had been injected. In other cases (110) on which we used one-tenth of 1 per cent nupercain with adrenalin, on both sides, between 10 and 30 c.c., according to the skill of the operator, some reported severe pain an hour or an hour and a half after the operation, while some of them had no considerable pain 18 hours after the operation. However, we know that the exact judgment of the pain in postoperative tonsillectomies is very hard to determine because the individual sensitiveness of the patient plays a greater role than in any other surgical operation.

There were no reactions similar to those we find after an injection of novocain, such as: fainting spells, and temporary anemia, except in one case a man of 30 years of age complained of a terrific headache at the time of the operation, which phenomenon is mentioned more often when nupercain is used as a spinal anesthetic. In another case, a short, stout man, about 32 years of age, complained of temporary shortness of the breath, which lasted only a few minutes after the injection and which probably can be attributed more to the adrenalin than to the nupercain. There was no postoperative reaction noted in any of these cases. The healing process did not show any difference in cases where novocain had been injected.

A very important factor in the technique is the exclusion of all traces of alkalin. Therefore, the syringe should not be boiled, or if boiled, should be cleaned with sterile distilled water.

The second point is the use of nupercain for topical application for operative purposes. The 2 per cent solution is one which gives a satisfactory anesthesia if applied on the mucous membrane of the nose, or in the pharynx or larynx. This percentage gives about the same result as the 10 per cent cocain. (My observations are based on more than 35 operations.)

Zeidler[10] notices injuries which proved to be inflammatory reactions in the larynx after using a higher percentage. Even with the 2 per cent solution he observed the same phenomena in one case. Another unpleasant occurrence with the use of 2 per cent nupercain for topical application is the hyperanemia, which, according to the German surgeons, can be sufficiently controlled with the addition of adrenalin. Klestadt[11], in Breslau, sounded the first warning in nose and throat practice against the careless use of a high percentage of nupercain. He observed three collapses after using 2 per cent nupercain at surgery of inferior turbinates.

I, personally, have never observed any unfavorable reaction after the use of nupercain topically, except that there is more bleeding than after the use of cocain, even with the increased addition of adrenalin, 1:1. The postoperative pain does not play a great role in nasal operations, therefore the advantage of the longer anesthesia is not of much importance. We find an excellent use of the drug at nasal operations, where shrinking is not necessary or even contraindicated (local adenoids, polyps, and inferior turbinates). Operative procedures in older children can be performed with nupercain with less danger than with cocain. It is very useful as a preliminary at tonsillectomies, combined with novocain, when we are dealing with a sensitive throat, which usually means gagging, used as a spray or for swabbing. It is just as effective as cocain in laryngeal examinations. At laryngeal operations, although it gives a satisfactory anesthesia, the cough cannot be sufficiently controlled. These are my observations and correspond with those of Ruedi, who had the same experience. Zeidler reports it to be a satisfactory anesthesia for laryngeal operations.

For diagnostic purposes in the nose, nupercain cannot substitute cocain because of the hyperemic effect. Ischemia is very important in making a correct diagnosis at nasal examinations and, therefore, the elimination of cocain for diagnostic purposes is not as yet possible. However, we call attention to the reduction of cocain with the use of the Gehse solution[12], 2 per cent cocain, one-third of 1 per cent phenol in saline, combined with adrenalin, which will give as excellent ischemia and anesthetic result as the 10 per cent cocain.

As to the use of nupercain for spinal and caudal anesthesia, which is beyond the scope of this paper, satisfactory reports appear by many general surgeons, its great advantage being the low specific gravity of the effective solution which can be made isotonic with the addition of saline.

Conclusions: In 1:1000 solution for local tonsillectomies it will give an effective anesthesia; but on account of the relative toxicity, we must be careful with its use. The lasting effect of nupercain has not definitely been proven.

For topical application as preliminary to tonsillectomies, it is very excellent and cocain can be completely eliminated. For topical application for operative purposes in the nose, where shrinking is not necessary, the drug will give excellent results. If shrinking is necessary, the cocain is still preferred, but in a reduced percentage. For diagnostic purposes in the nose, it is inefficient.

BIBLIOGRAPHY.

1. KEYES AND MCLELLAN: *American. Jour. of Surgery,* 1930.
2. BECK: Annals of Otolaryngology, 1930.
3. MISCHER: Narcose and Anesthesia, 1929.
4. ULLMAN: Narcose and Anesthesia, 1929.
5. LIPSCHITZ and LAUBENDER: Klin. Wschr., 1929-30.
6. RUEDI: Schweiz. Med. Wschr., 1930.
7. SIEBNER: Der Chirurg., 1929.
8. FREUND: Klin. Wschr., 1929.
9. ALBERSHEIM: Zschr. f. Laryng., 1930.
10. ZEIDLER: Wiener Klin. Wschr., 1930.
11. KLESTADT: Munch. Med. Wschr., 1929.
12. LASZLO: Annals of Otolaryngology, 1931.

124 East 84th Street.

IMPROVED POWDER BLOWERS FOR APPLYING IODIN POWDER (SULZBERGER).*

Dr. M. D. Lederman, New York.

In applying iodin powder (Sulzberger) in aural and nasal infections, I have found these improved powder blowers more satisfactory than the usual blowers on the market. At times one meets with cases where it is difficult to apply the powder into small perforations of the membrana tympani, especially when these are located at the superior margin of the drum.

The instrument here presented is adjustable so that the powder can be satisfactorily placed in any portion of the drum. The adjustable extension is two inches long, with a sixteenth of an inch bore, and with gentle air pressure one can introduce powder into very small perforations. Too much air pressure will divert the powder from the cavity. The advantage of the dark-colored bottle blower is that one has a supply on hand ready for continued use, without the necessity of reloading for each treatment. Iodin powder should be kept in a dark container to avoid deterioration.

The adjustable instrument permits easy applications of the powder in nasal suppurations, after the secretion has been thoroughly removed, by the following technique: *1.* Spraying the nasal cavities with 1 per cent solution of ephedrin sulphate solution or adrephin solution; then waiting 10 minutes. *2.* Gentle suction to remove as much secretion as possible. *3.* Any suitable alkalin spray to remove secretion still remaining. *4.* It is usually advisable to precede the application of the powder with some local anesthetic, *i. e.*, a weak solution of cocain muriate, 0.5 to 1 per cent, in the form of a spray: novocain, 2 per cent solution, or nupercain, 2 per cent. This precaution prevents the smarting sensation which at times occurs for a short time as the iodin vapor is given off. Some patients are more sensitive than others, though I have applied the powder at times without any anesthesia. In a few instances I have applied strong powder in atrophic rhinitis, after the crusts have been removed, without local anesthesia, with pleasant results. *5.* Application of the weak (1 per cent) iodin powder (Sulzberger) through adjustable

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blower. First apply the powder to the space between the middle turbinal and outer wall. Then to the space between the middle turbinal and nasal septum. Lastly, covering the anterior surface of the middle turbinal with a coating of the powder.

If a surgical opening has been made into the antrum and secretion persists, the small adjustable extension can be introduced into the opening and the powder can be applied directly into the antrum.

The powder may be used with satisfactory results after any surgical intervention in the nose, after bleeding has been controlled. The patient should be cautioned not to blow the nose forcibly for an hour following its introduction, so that its therapeutic effect, due to the rapid elimination of the nascent iodin, may follow. Accumulated secretion may be wiped away, as the effect of the powder promptly stimulates osmosis. One need not fear retention of secretion, as one of the great advantages of this preparation is that it

dissolves very readily in the presence of moisture; *not being a chemical entity,* it gives off its iodin content with great rapidity.

The liberation of the iodin vapor is promptly felt by the patient in the region of the nasal sinuses in a short time. Suffusion of the conjunctiva with lacrimation and marked increase in nasal secretion is observed. This reaction soon subsides, and the individual feels the nasal cavities clearer, and the symptom of obstruction much relieved.

One should permit the acute inflammatory stage to subside before the powder is employed.

The other blowers exhibited may be used where the perforations of the membrana are of fair size, and the one with the metal shovel can be used for nasal treatments as well, as its bore is smaller than the hard rubber one. Furthermore, it can be sterilized.

These instruments are made by George P. Pilling & Son Co., Philadelphia, Pa.

106 East 84th Street.

International Digest of Current Otolaryngology.

Editor:
DR. MAXWELL FINEBERG, St. Louis.

Collaborators:
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

Ayres and Anderson, in the *Jour. A. M. A.*, Aug. 15, 1931, write on Dermatitis Medicamentosa Due to Ephedrin. They report two cases of this following nasal use of the drug. Knowledge that ephedrin can produce such cutaneous manifestations may be of value in determining the cause of obscure eruptions about the nose, face and elsewhere.

Having used stock respiratory vaccines in one thousand cases, Forbes in the American Journal of Clinical Pathology for September, 1931, favors this procedure especially in the upper respiratory infections of children. He recommends a heavy bacterial emulsion and adequate dosage. He reports many favorable results following only one injection. ROSENBERGER.

The Sixth International Congress of Genetics will be held Aug. 24-31, 1932, in Ithaca, N. Y. A request has been received from Holland that some publicity be given this subject with the hope of stimulating interest in the several international meetings that are to be held within the next year.

Wirth, in the Zeit. f. H., N. u. O., 28:189:1931, describes his experiments on local selectivity of tonsil extract injections. He worked with numerous rabbits, attempting to reproduce the disease with which the donor suffered. In fifteen cases of joint arthritis his results in selective localization were positive nine times; questionable three times, and negative three times. In three cases of sepsis he had two positives and one negative. In six cases of nephritis he had three questionable results and three negative results. In three cases of endocarditis there was no selective localization susceptibility to tonsil extract. KELEMEN.

Leichsenring, in the Zeit. f. H., N. u. O., 28:220:1931, describes the treatment of allergic diseases by alcohol injections in the nasal mucous membrane. He injected one or two c.c. of 40 to 60 per cent alcohol in the presenting part of the inferior turbinate directly opposite the septum. A preliminary cocain anesthesia is applied; there is an immediate reaction which, however, disappears in a few hours. The author reports good results in 80 per cent of his cases of hay fever and vasomotor rhinitis. Many of his cases were improved for well over a year, but in hay fever he believes one should re-inject every year. KELEMEN.

In an editorial in the *Jour. A. M. A.*, May 30, 1931, there is a brief discussion of Intra-Auricular Puncture. Physiologists and clinicians admit that almost miraculous results have occurred from intra-cardiac puncture. With a patient on the operating table, whose heart has suddenly stopped, there is no time to consider the fine points of the value of a procedure that will bring back to life a seemingly dead patient. For this reason every surgeon should be able to act skillfully and quickly when the emergency occurs.

This particular editorial has to do with the added advantages of intra-auricular as against intra-ventricular puncture. Many drugs have been used; the following list seems to have some advocates: Metrazol, ether caffein, dextrose, sodium thiosulphate, camphor, digitalis, strophanthin, strychnin and ephedrin. Intra-auricular puncture is performed by inserting a curved needle into the third interspace at the right sternal margin, directing the point downward and toward the median line. In children the distance to the right auricle is two inches or less; in adults, four inches or less.

At biweekly periods for almost two years, eighteen children (Rochester, Minn.) from two to fourteen years of age had a total of four hundred eighty-six nasopharyngeal swab cultures made. Of this number 44 per cent were positive for streptococcus hemolyticus and streptococcus viridans was practically constantly present. During this period ninety-eight upper respiratory infections occurred with streptococcus hemolyticus present in 50 per cent. The mere presence of streptococcus hemolyticus is considered inconsequential but its presence with a high percentage of organisms may be of etiological significance. This laborious and interesting work is reported by Helmholz in the American Journal of Diseases of Children for August, 1931. ROSENBERGER.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Meeting of May 8, 1931.

Bilateral Labyrinthine Hemorrhage in a Case of Aplastic Anemia. Dr. M. Rosenbluth.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Two Unusual Cases of Labyrinthitis. Dr. Chas. M. Griffiths.

(Published in full in this issue.)

DISCUSSION.

DR. M. D. LEDERMAN: I wish to express my admiration for and appreciation of the very erudite paper by the two gentlemen, though I do not feel qualified to speak upon the experimentation and the interesting phenomena exhibited here. When one realizes how difficult it is to arrive at a conclusion in regard to the human being in these tests, we cannot but admire the painstaking efforts of the men who were kind enough to present the question this evening.

I would like to say a few words on the case presented by Dr. Rosenbluth. I had the opportunity of seeing this case rather late in the course of the disease, and in listening to the history and remarks made by Dr. Rosenbluth, I feel that there are two points in the case which must not be overlooked: the fact of the existing condition of the patient previous to the destruction of the hearing. While the case was no doubt one of plastic anemia and hemorrhage of both labyrinths, we must not overlook the fact that syphilis was also a very prominent factor. If I recall the history correctly, there were no distinct symptoms of nausea or vomiting. The loss of hearing was very sudden. The fact that he had had two or three courses of arsenical treatment may have played an important part in the destruction of audition from a chemical standpoint; but I feel that the underlying cause was in the syphilitic infection. The response to the iodin powder (Sulzberger) treatment was rather striking, but on account of the hemorrhage and proneness to bleed we thought that discretion was the better part of the management and applied the dry method of treatment for the existing granulation tissue. That acted very satisfactorily.

DR. MARVIN JONES: If I understood the history correctly, this man had a pair of acute ears, which were allowed to rupture spontaneously. I would like to know if that procedure is usually followed. The next point is the fact that the sudden loss of hearing in the two ears occurred simultaneously—a rare condition outside of a cerebral lesion. It is conceivable that the hemorrhage would have that bilateral effect, but one could have a hemorrhagic condition, particularly of the fourth ventricle, which might account for the condition. The fact that there was no spontaneous nystagmus would mean that both ears would have been affected at the same time. It is in the bounds of possibility, but hardly probable, that such a thing could have occurred.

We have had the usual classical presentation from Dr. Pike. Very few of us are mentally capable of even following his presentation, let alone discussing it, but it is always a pleasure and stimulation to hear him.

DR. JOHN GUTTMAN: With regard to Dr. Rosenbluth's case, I would like to call attention to the fact that the symptom-complex which is known as "Meniere's disease," consisting of deafness, dizziness, nystagmus and disturbance of equilibrium, was originally observed and described by Meniere in a 17-year-old girl, who was suffering from pernicious anemia; and at the post-mortem examination the inner ear was found filled with blood. Dr. Rosenbluth's case, however, has many peculiar features; for instance, the lack of spontaneous nystagmus. The offered explanation that the bleeding occurred spontaneously in both labyrinths simultaneously is not very convincing.

In regard to Dr. Pike's presentation, I am sure that he noticed an occasional spontaneous nystagmus in anesthetized animals. In the literature cases are mentioned where in an anesthetized man spontaneous nystagmus was observed. I would like to know what explanation he has to offer for this phenomenon. Is this nystagmus of central or peripheral origin?

The quantitative measurement of the reaction of the vestibular nerve in man was suggested by Breuning and others, but it did not prove very satisfactory. The recording of the amplitude frequency, etc., in animals, as was done by Dr. Northington, was probably much more difficult.

DR. L. M. HUBBY: I was very much interested in what Dr. Pike had to say, and two points in Dr. Northington's experiments on cats attracted my attention as I followed them in his charts. One was that the destruction of one labyrinth was followed by increased response and irritability in the other both to rotation and to the caloric test. With the human being our experience has been the other way. There has been a decrease of irritability on the other side after the destruction of one labyrinth.

Another thing was that the destruction of the cerebellum or an operation beyond the labyrinth increased apparently the labyrinthine irritability, *i. e.*, increased the length of the nystagmus and also the irritability to the caloric. Neuman called such a nystagmus "enduring nystagmus." The symptom is due to the loss of restraint and control of the higher centers.

Dr. Rosenbluth's case was a very interesting and unusual one. As to the mechanism of how it all occurred, it seems to me that in the first place there was an infection of both middle ears. He came into the hospital with a temperature of 103°. On account of the condition of the blood there was considerable bleeding on rupture of the drums. I think the rupture of the drums was due to the infection and not to the extravasation of blood in the middle ears. After the otitis had gone on for a period of time—fully a month—the ear condition got worse, granulations appeared, and then one night, suddenly he lost the hearing in both ears. If the labyrinthine condition was produced by what was occuring in the middle ears, it might be due to two causes: one being the increase in intralabyrinthine pressure from the blocking and granulations in the middle ears pressing upon the labyrinthine windows, and also increased reflexly through irritation of the tympanic plexus of nerves. This increased intralabyrinthine pressure occurring in one who already had a tendency to bleed, would account for the simultaneous hemorrhage in both internal ears.

Another point, in destruction of both labyrinths at about the same time there are no afferent impulses from them to the vestibular centres, therefore no reflex of nystamus, vertigo, etc. This has been shown to be true in animal experimentation.

Considering the fact that the patient had such a severe anemia and such a succession of hemorrhages elsewhere, I believe that Dr. Rosenbluth's diagnosis of hemorrhage in both labyrinths is more probable than that of double diffuse serous labyrinthitis.

DR. S. BERNARD WORTIS: I want to say a word concerning the paper by Dr. Pike and Dr. Northington. Dr. Pike for many years has been paving the way to a better understanding of the problems of neurophysiology, and particularly phenomena concerned in convulsions and nystagmus.

At the Bellevue Hospital Laboratory of Experimental Neurology we have been confirming and extending some of Dr. Pike's work in this field. At Bellevue we have also attempted to correlate our clinical judgments, in the light of these newly acquired facts.

The combined experimental and clinical approaches to the problems of epilepsy and nystagmus will, I believe, yield us most valuable data. We also use the cat as our experimental animal and employ camphor monobromid as our standardized convulsant. Also as an "offshoot" of our studies in epilepsy we have been interested in some of the problems of nystagmus.

Any study dealing with nervous tissue must take cognizance of the problems of nerve irritability. We have shown at Bellevue that brain trauma and certain drugs increase the irritability of nerve tissue. These findings are in agreement with work by other investigators who have utilized the method of electrical stimulation of the central nervous system.

Magnus and his coworkers of the Utrecht School have recently demonstrated the roles of the various components comprising "reflex posture mechanisms." The labyrinth has been shown to comprise a very important centre for the maintenance and alteration of posture of the organism. We must not forget that there is a cerebral component in nystagmus. Often in decerebrated animals, head and conjugate eye deviation are obtained following rotation tests, but no quick nystagmus component results. This also obtains during deep surgical anesthesia. The cerebrum therefore must represent an additional essential factor in the production of nystagmus. Anesthesia also prevents convulsion phenomena in laboratory animals.

All this information is of very practical importance to the clinician, in the following conditions: 1. Inflammatory or toxic lesions affecting the labyrinth in people give rise, as you well know, to nystagmus and extreme vertigo. 2. In cases where the labyrinth has been completely destroyed by disease, or where the conducting ability of the eighth nerve has been completely obliterated no spontaneous nystagmus obtains, and none is elicitable by the Barany tests. These persons usually do not have vertigo.

This field of medical knowledge is the meeting place for neurologists and otologists. Here both specialists must unite to the procurance of better clinical diagnostic judgment in the realm of brain disease and injury.

One of the most difficult diagnostic problems presented to the neurologist is the differential diagnosis of right frontal, from a left cerebellar lesion. It is in this field that the otologist can render aid; by determining by both the Barany and caloric tests the functional activity of the labyrinthine reflexes.

Let me add one more word concerning the clinical observations of the different types of nystagmus. Usually the nystagmus which obtains in cerebellar diseases is of a coarse type, whereas that dependent upon labyrinthine disease has a very rapid, very fine quality.

Again let me thank Dr. Pike and Dr. Northington for their excellent presentation of a most difficult and most important problem.

I saw Dr. Rosenbluth's case, when or where I do not exactly remember. When I looked into the ear I saw an enormous polyp sticking out. In the history of the case, if a man develops infection you don't get polyps in an acute ear, and at that time you cannot touch or move him. Is it not possible that he had a purulent labyrinthitis that finally led to destruction? The man also had syphilis, which possibly explained it.

Dr. E. M. Josephson: Dr. Pike and Dr. Northington are to be congratulated on their very interesting presentation. The experiments which Dr. Pike presented bring to mind the work of Magnus and de Kleyn on animals decerebrated by section opposite the corpus striatum. They demonstrated head-position body muscles reactions, which they termed neck and head reflexes, in these animals, manifesting themselves by turning of the limbs on the affected side in the same direction in which the head is turned, which reactions they ascribed to the vestibulocerebellar apparatus. Similar reactions have been described by myself and by Brouwer, Bohme and Weiland and others in disease and tumor formation in the brain stem or pressing upon it. The pure vestibular reflexes of lower animals do not manifest themselves so openly in the human because a higher organization of the central nervous system has resulted in a displacement of the role of the vestibular function in the static and kinesthetic sense to a role secondary to the summation of impulses from numerous senses, as was first expressed by Loeb.

It was asked, by one of the discussors, of what clinical value was the work presented this evening. It has a distinct and high value in explaining some of our clinical and neurologic findings. It helps to explain the head-body muscle reaction above mentioned, the past-pointing on turning of the head in cases with irritation lesions of the brain stem. The cat experiments, of which Dr. Pike showed slides and illustrations, can be interpreted as indicating one damage which is essential to secure predominance of the action of vestibular and other impulses constituting the static and kinesthetic sense in a more highly organized nervous system. The position in which the cat holds its head, the sound ear rotated upward is probably due to predominance of the function of the vestibular apparatus on that side, though there is not enough data at

hand to make such an inference absolutely conclusive. Of special interest is Dr. Pike's mention of the cat's reaction to sound, in response to sound coming from above, the cat turned to the side of the sound ear. This helps impress upon us a fact which most of us overlook, that there is an intimate though not yet thoroughly elucidated inter-relation between auditory and vestibular function which one might well suspect from the extreme juxtaposition of the two organs. In the human this relationship seems to take the form of an inverse ratio, high auditory efficiency is associated generally with low irritability of the vestibular apparatus, and low auditory efficiency with higher irritability of the vestibular apparatus, until the function of both structures is destroyed.

Not only is this work of Dr. Pike's of interest in connection with the equilibration mechanism in the adult as above mentioned and in the understanding of such conditions as torticollis spasticus and others, but it throws additional light on the possibilities of understanding of conditions such as spasmus nutans occurring in infancy before the full development of the central nervous system, some cases of which have recently been reported in the German literature as being associated with vestibular lesions.

I wish to ask Dr. Pike if he does not feel that covering the eyes of the animals before testing by rotation might not in some manner affect the results. Does he not regard the visual component of the sense of equilibrium of sufficient influence to alter the results? Fischer and Wodak and I, working independently, have demonstrated the marked influence of the oculovestibular past-pointing reactions, and many other workers have demonstrated the importance of the ocular element in the testing of vestibular reactions.

In connection with Dr. Rosenbluth's case, I might mention that in the course of studies on progressive deafness, I have found it possible to reduce the hearing almost to total deafness by pressure upon the carotid artery on the side tested, thus creating a local ischemia.

DR. H. B. BLACKWELL: As chairman of this meeting I would like to express approval of the conservative attitude which Dr. Griffiths so successfully adopted in his two cases, and would also ask Dr. Rosenbluth concerning the dosage and variety of arsenic used in his case; the Section is fortunate in hearing the highly interesting and scientific papers contributed by Dr. Pike and Dr. Northington. Dr. Pike's observations concerning the dual function of the labyrinth and cerebellum are very stimulating and possibly account for the atypical phenomena which otologists sometimes observe in patients with complications affecting these structures.

Vestibular Tests on Animals with Experimental Lesions of the Nervous System, with Lantern Slides. F. H. Pike, Ph.D., and Dr. Page Northington.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION OF OTOLARYNGOLOGY.

Meeting of April 10, 1931.

Dr. John Brown, President, Presided.

Laryngeal Emergencies in Infancy and Childhood. Dr. Kenneth Phelps.

DR. IRVINE MCQUARRIE (by invitation) expressed his thanks for the invitation to attend this meeting and to discuss Dr. Phelps' paper. He said he enjoyed very much Dr. Phelps' review of this subject, which was covered so completely that there was very little a pediatrist could contribute in addition. Pediatrists do see these patients at an early period very often and it is essential that they know all they can about these emergencies in the trachea in order to co-operate intelligently with the laryngologist. One thing which Dr. McQuarrie said interested him particularly was Dr. Phelps' emphasis upon the importance of the direct examination, which even pediatrists are coming to do in cases of laryngeal diphtheria in which they can suck out the loose membrane. He himself had been in three different clinics where that had been done routinely with great benefit to the patients. In 40 per cent of the cases where it is done promptly it makes intubation unnecessary. Also in connection with what Dr. Phelps had said about streptococcic laryngitis, Dr. McQuarrie agreed that intubation is contraindicated and tracheotomy should be done early. He had seen, in two recent small epidemics, the occurrence of this condition, in which children from 15 months to 2 years, had a very severe purulent laryngitis. Early intubation was done at first, but they all died. Then seven of the later ones were tracheotomized when the obstruction appeared to be severe enough to demand treatment. When tracheotomy was done and was followed by frequent suction carried out by a special nurse, most of the babies were saved.

Dr. McQuarrie said he would like to say a word about laryngismus stridulus, which is one manifestation of infantile tetany. In pediatric literature this means the type that is due to spasm or increased irritability of the muscles generally. These children have an inspiratory crow that one can hear all over the room. The explanations which Dr. Phelps gave included spasm, of course, and Dr. McQuarrie believed that spasm is one feature at least, because it is so quickly relieved, along with other spasmodic signs when calcium therapy is begun. Although the calcium metabolism is disturbed in this condition it is not known whether spasm is due to a lower calcium level of the blood or whether it is a primary disturbance in the brain cells themselves. It has been found that there is increased hydration of the brain cells in tetany, which may contribute to the hyperirritability usually attributed alone to a lowered calcium ion concentration in the tissues.

Dr. McQuarrie referred to two conditions which had recently been seen in the Pediatric Ward of the University Hospital, which were not alarming but were interesting to the staff. One was hypoplasia of the mandible. The baby looked like "Andy Gump," and had marked cyanosis when on its back. If placed on the face with a pillow under the chest, these babies will breathe very well. He had seen two such cases of micrognathia. The other was a case of real cretinism, which was lost before thyroid extract had taken effect. This baby had not only a very large tongue but at autopsy was found to have a swelling of the larynx and a tremendous thickening of the diaphragm, due to water retention in its tissues. The baby had several cyanotic attacks that would make one think of laryngeal obstruction.

DR. C. E. CONNOR said he had nothing to add except to second what Dr. Phelps had said about the ease of laryngoscopy in small children. It permits

one to look into the larynx and tell what the difficulty is; there is nothing which enables one to proceed with therapy in as satisfactory manner as looking in directly with the laryngoscope.

Dr. Kenneth Phelps (in closing) said he wished to thank Dr. McQuarrie for his discussion and he thought Dr. McQuarrie could tell the otolaryngologists more about the spastic side of laryngismus stridulus than they could tell with directly bronchoscopy alone. He felt that when one looked through a tube he limited himself to just what he could see through that tube, and was inclined to become rather narrow-minded, rather than to take in the whole condition of the patient.

Acute and Chronic Ethmoiditis. Dr. Fred J. Pratt.

DISCUSSION.

Dr. Paul Berrisford said he would like to ask three questions: 1. Where is this pain? 2. What is the character of the pain? 3. How do you differentiate this pain from the pain originating in other sinuses, or what in the history would lead one to believe that it is the ethmoid?

Dr. Pratt said it was difficult at times to find a definite area of pain from the ethmoids because if other sinuses are involved the pain will be all over the head. He felt that one would have to rule out the other sinuses. As a rule if the anterior ethmoids are involved, the posterior are involved as well, and the frontal, which is rarely an ethmoid, is generally also involved. As to the area of pain, Dr. Pratt said there are different rules; usually the pain is over the cheek for that antrum, but this year the patients complain of pain over and around the eye. The frontal pain is over the forehead, the anterior ethmoid naturally would be over the bridge of the nose, and the posterior ethmoid would be deeper in the head and the sphenoid pain in the back of the neck. Patients cannot always tell the doctor just where the pain is, and one may have to talk to them a long time to get any definite place as to their headache. Dr. Pratt said he would classify the acute ethmoiditis as the ordinary colds in which there is severe headache. As to the justification for surgery in the subacute stage, Dr. Pratt said he would justify surgery in pain that is not relieved with local treatment; pain that continues in spite of local treatment over a week or more. These patients must have relief in some way; they must have drainage and aeration.

Dr. F. N. Knapp asked, in a patient with a cavity on the right side and the septum deflected to the left, why is it one usually finds pus in the concavity?

Dr. Pratt said, because the patient has a compensatory hypertrophy of the turbinate on that side, which swells and prevents drainage.

Dr. Knapp said, then it is the turbinate and not the septum?

Dr. Pratt stated that, primarily it is the septum.